U.S. Department of Labor

Office of Administrative Law Judges
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Issue Date: 09 February 2004

In the matter of
Ruth Frasure, Widow of
Scott Frasure, Deceased,

2003-BLA-05512

Claimant

v.

Hope Mining Company, Inc., and
Old Republic Insurance Co.
Employer/Carrier

and

Director, Office of Workers' Compensation Programs
Party-in-Interest

APPEARANCES:

On Behalf of Claimant:
Stephen A. Sanders, Esq.
Appalachian Citizens Law Center, Inc.
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On Behalf of Employer:
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BEFORE:
Daniel F. Solomon
Administrative Law Judge

DECISION AND ORDER DENYING MODIFICATION AND DENYING SURVIVOR BENEFITS

JURISDICTION

This proceeding arises from a claim for benefits under the Black Lung Benefits Act, 30 U.S.C. § 901 *et seq* (the Act). The Act and implementing regulations, 20 CFR Parts 410, 718, 725 and 727, provide compensation and other benefits to living coal miners who are totally disabled due to pneumoconiosis and their dependents, and surviving dependents of coal miners whose death was due to pneumoconiosis. The Act and regulations define pneumoconiosis, commonly known as black lung disease, as a chronic dust disease of the lungs and its sequelae, including respiratory and pulmonary impairments, arising out of coal mine employment. 30 U.S.C. § 902(b); 20 CFR § 718.201 (2003). Preliminarily, it should be noted that this case consists of two consolidated claims: (1) A modification claim originally filed by the miner, Scott

Frasure, on July 2, 2001, and ultimately pursued by his surviving spouse, Ruth Frasure, administrator of the estate; and (2) A survivor claim filed by Ruth Frasure on December 18, 2001.

MODIFICATION CLAIM

Claim History

Claimant, Scott Frasure, originally filed an application for federal black lung benefits on May 29, 1984. (DX 55). In his application, he indicated that he had worked in or around coal mines for fifteen years, six months, and that he stopped working on January 27, 1984 as a result of being "laid off" and then "unable to go back to work." (DX 55). He also indicated that he had emphysema and "difficulties breathing dust." (DX 55). Although the Department of Labor (DOL) authorized a medical examination of Mr. Frasure, he refused to undergo the exam because "[his] doctor and lawyer advised [him] that [he] did not have Black Lung and [he had been] to five doctors." (DX 55). On September 18, 1994, DOL determined that Mr. Frasure had abandoned his claim. (DX 55).

Mr. Frasure filed a second application for federal black lung benefits on June 28, 1993. (DX 1). On the second application, Mr. Frasure indicated that he had worked in or around coal mines for fourteen years, six months and that he stopped work in June, 1984, due to an on-the-job back injury. (DX 1). He also indicated that his last coal mine employer was Hope Mining Company in Swamp Branch, Kentucky. (DX 1). Mr. Frasure's form describing his coal mine work and other employment, filed July 17, 1993, indicated that he had worked three to five days per week at Hope Mining Company as a truck driver from 1971 to 1984. (DX 6). He would sit for 7.25 hours per day and stand for fifteen to twenty minutes per day. (DX 6). He would not do any crawling, lifting, or carrying. (DX 6). On March 15, 1994, the District Director awarded benefits with regard to Mr. Frasure's second application for benefits under the Act. (DX 53). The Employer appealed. (DX 54).

Mr. Frasure's claim proceeded to the Office of Administrative Law Judges where a formal hearing was held before Administrative Law Judge Paul H. Teitler on October 19, 1995. (DX 100). Judge Teitler awarded benefits in a decision issued April 2, 1996 (DX 100). The Employer appealed. (DX 105). On May 23, 1997, the Benefits Review Board issued a decision affirming in part and vacating in part the award of benefits. (DX 105). The case was remanded for further consideration. (DX 105). On remand, Judge Teitler again awarded benefits in a decision issued November 7, 1997. (DX 107). The Employer appealed once again. (DX 113). On September 13, 1999, the Board issued a decision affirming in part and vacating in part the award of benefits. (DX 113). The case was again remanded for further consideration. (DX 113). On remand, Judge Teitler found that the Claimant had failed to establish the existence of pneumoconiosis. (DX 116). Accordingly, he denied benefits without considering the total disability issue in a decision issued April 20, 2000. (DX 116). On May 17, 2000, Claimant filed a Notice of Appeal, which was acknowledged on May 24, 2000. (DX 117, 118). On September 19, 2000, the Board issued an Order permitting the withdrawal of John Earl Hunt, Esquire as the Attorney of Record for Claimant. (DX 122). On October 23, 2000, Steven Sanders entered a

Notice of Appearance of Counsel for Mr. Frasure. (DX 125). On May 24, 2001, the Board issued a decision affirming the ALJ's denial of benefits. (DX 130).

Thereafter, in a letter addressed to the District Director and dated July 2, 2001, counsel for claimant requested modification of the Board's denial of benefits. (DX 131). Specifically, the letter stated that Claimant submits that "the denial of his claim contains mistakes of fact." (DX 131). The letter also stated, "[f]urther, his condition has worsened." (DX 131). In a letter dated August 6, 2001, the District Director stated that the correspondence would be treated as a request for modification because the previous claim had been denied less than one year prior to the correspondence. (DX 133). On August 28, 2001, counsel for Claimant submitted evidence to the District Director regarding the request for modification, including a deposition of Dr. Ayesha Sikder taken August 2, 2001 and records from the Highland Regional Medical Center. (DX 134). On October, 29, 2001, Claimant died. (DX 152). A decision had not yet been rendered regarding his request for modification. On November 20, 2001, Mr. Frasure's spouse, Ruth Frasure, was appointed as administrator of the estate. (DX 138). Ruth Frasure proceeded to pursue Mr. Frasure's modification claim. (DX 141). On December 16, 2002, the District Director issued a Proposed Decision and Order Granting Claimant's Request for Modification. (DX 143). Specifically, it stated that "a review of the evidence of record establishes the miner had a material change in condition prior to his death which occurred on October 29, 2001." (DX 143). On December 20, 2001, Employer appealed and requested a hearing. (DX 144). On February 21, 2003, the matter was referred to the Office of Administrative Law Judges. (DX 177).

The case was subsequently assigned to me on April 4, 2003. On June 25, 2003, Employer filed a Motion to Strike the deposition testimony of Dr. Sikder taken on August 2, 2001. In support of its Motion to Strike, Employer asserted the following series of events: (1) On August 6, 2001, correspondence acknowledging Claimant's request for modification was served upon Claimant's counsel and Employer's former counsel; (2) Claimant's counsel took the deposition of Dr. Sikder on August 2, 2001 before this correspondence was issued; (3) Dr. Sikder's deposition testimony was taken without the Employer "being present and having an opportunity to cross-examine." In sum, the Employer alleged that this series of events "operates a denial of . . . due process rights of law" and warrants the striking of Dr. Sikder's deposition testimony. In its Motion to Strike, the Employer also notes that it contacted Dr. Sikder's office several times to attempt to schedule her deposition on cross-examination but that Dr. Siker was not compliant.

Claimant responded to Employer's Motion to Strike by asserting that: (1) On July 2, 2001, notice that the deposition would be taken on August 2, 2001 was mailed to the attorney who had been representing the Employer before the BRB; (2) Also on July 2, 2001, counsel for Claimant mailed a letter to the District Director requesting modification and stating that he intended to submit additional evidence in support of such a request; (3) At no time prior to Dr. Sikder's deposition did the Employer make any objection; (4) The deposition was submitted to the District Director on August 28, 2001 and mailed to Employer's counsel, who did not object at that time. In sum, Claimant argued that Employer was not denied due process, but rather failed to avail itself of the opportunity to cross-examine Dr. Sikder. Accordingly, Claimant argues, Dr. Sikder's deposition testimony should not be stricken.

On August 5, 2003, I conducted a telephonic hearing on Employer's Motion to Strike during which I denied the Motion. On August 8, 2003, Employer requested a subpoena directing the appearance of Dr. Sikder at the upcoming hearing. A subpoena was thereafter issued. On August 19, 2003, a formal hearing on the claim was held in Prestonburg, Kentucky. The preliminary matter of Dr. Sikder's testimony was addressed. Attorney for Claimant advised that he was never sent a copy of the subpoena directing the appearance of Dr. Sikder. (Tr. 6). Attorney for Employer advised that Dr. Sikder would not be appearing at the hearing due to exigent circumstances and that she had agreed to give a deposition post-hearing. (Tr. 6). Attorney for Claimant objected to the post-hearing deposition. (Tr. 8). He stated that he received a letter on August 15, 2003, only four days before the formal hearing, which advised that Attorney for Employer intended to call Dr. Sikder as a witness. (Tr. 9). Upon receiving the letter, Attorney for Claimant called Dr. Sikder's office and learned that she had not been notified that she was to appear as a witness at the hearing. (Tr. 9). Thereafter, one day before the hearing, Attorney for Claimant heard from Dr. Sikder's office that she had just received a subpoena but could not attend the hearing due to exigent circumstances. (Tr. 9). Ultimately, I struck the subpoena that I had issued to Attorney for Employer and rescinded his authority to take the deposition post-hearing. (Tr. 14). 20 C.F.R. 725.455.

Also at the hearing, Director's Exhibits Nos. 1 through 177 were admitted into evidence (Tr. 22) and Employer's Exhibits Nos. 1 through 6 were admitted into evidence (Tr. 26).[1] No Claimant's Exhibits, ALJ Exhibits, nor Joint Exhibits were admitted into evidence.

Material Change in Condition

Any time within one (1) year of a denial or award of benefits, any party to the proceeding may request a reconsideration based on a change in condition or a mistake of fact made during the determination of the claim. 20 C.F.R. § 725.310. However, after the expiration of one (1) year, the submission of additional material or another claim is considered a duplicate claim which will be denied on the basis of the prior denial unless the claimant demonstrates a material change in conditions under the provisions of 20 C.F.R. § 725.309 as interpreted by the Benefits Review Board and Federal Courts of Appeal. Under this regulatory provision and according to the Court of Appeals for the Sixth Circuit in ***Sharondale Corporation v. Ross***, 42 F.3d 993, 997-98 (6th Circuit 1994):

> [T]o assess whether a material change is established, the ALJ must consider all of the new evidence, favorable and unfavorable, and determine whether the miner has proven at least one of the elements of entitlement previously adjudicated against him. If the miner establishes the existence of that element, he has demonstrated, as a matter of law, a material change. Then, the ALJ must consider whether all of the record evidence, including that submitted with the previous claims, supports a finding of entitlement to benefits.

I interpret the ***Sharondale*** approach to mean that the relevant inquiry in a material change case is whether evidence developed since the prior adjudication would now support a finding of

[1] References to "DX" are exhibits of the Director. References to "EX" are exhibits of the Employer.

an element of entitlement. The court in ***Peabody Coal Company v. Spese,*** 117 F.3d 1001, 1008 (7th Cir. 1997) put the concept in clearer terms:

> The key point is that the claimant cannot simply bring in new evidence that addresses his condition at the time of the earlier denial. His theory of recovery on the new claim must be consistent with the assumption that the original denial was correct. To prevail on the new claim, therefore, the miner must show that something capable of making a difference has changed since the record closed on the first application.

On April 20, 2000, Judge Teitler denied Frasure's claim because he had failed to establish the existence of pneumoconiosis pursuant to 20 C.F.R. § 718.202. (DX 116). Accordingly, Judge Teitler denied benefits without considering the issue of total disability. (DX 116). The Board affirmed the denial of benefits in a Decision and Order dated May 24, 2001. (DX 130). As a result, to demonstrate that a change in condition has occurred since the denial of his prior claim, Frasure must prove, based on evidence developed since May 2001, the threshold issue of existence of pneumoconiosis pursuant to 20 C.F.R. § 718.202. I find that Claimant has failed to do so and, therefore find that Claimant has failed to establish a material change in his condition since his last application of benefits was denied.

Mistake in Determination of Fact

Although I find that Claimant has failed to establish a change in condition, I will re-open the record in this case to admit the evidence submitted by Claimant and Employer to determine if a mistake of fact was made in determining that Claimant did not suffer from coal workers' pneumoconiosis. The United States Supreme Court held in ***O'Keefe v. Aerojet-General Shipyards***, 404 U.S. 254 (1971), that the Deputy Commissioner is vested with a broad discretion to correct mistakes of fact, whether demonstrated by wholly new evidence, cumulative evidence, or merely further reflection on the evidence initially submitted. *Id*. at 246. Whether this claim should be re-opened for modification is discretionary, as the Act and 20 C.F.R. § 725.310 provide that the fact finder "may" reconsider the terms of an award or denial of benefits. The Sixth Circuit in York v. Director, OWCP, 82 F.3d 419 (6th Cir. 1996), considered the breadth of a court's discretion to open a case for modification. The court held:

> In deciding whether to reopen a case under 33 U.S.C. § 922, a court must balance the need to render justice against the need for finality in decision making. General ***Dynamics Corp. v. Director, OWCP***, 673 F.2d 23, 25 (1st Cir. 1982). As the Supreme Court stated in ***Banks v. Chicago Grain Trimmers Ass'n***, 390 U.S. 459, 464 (1968), the purpose of 33 U.S.C. § 922 is to permit a district director to modify an award when there has been "a mistaken in determination of fact [which] makes modification desirable in order to render justice under the act." *See also* ***Blevins v. Director, OWCP***, 683 F.2d 139, 142 (6th Cir. 1982). An allegation of mistake should not be allowed to become a back door route to retrying a case because one party thinks he can make a better showing on the second attempt. ***McCord v. Cephas***, 532 F.2d 1377, 1380 (D.C. Cir. 1976). As the Supreme Court made clear in its reference to the legislative explanation for the 1934 broadening of

the grounds for reopening under § 22, the basic criterion is whether reopening will "render justice" under the Act. ***O'Keefe***, 404 U.S. at 265.

In his November 7, 1997 Decision and Order on Remand Awarding Benefits, the administrative law judge (ALJ) found that Claimant failed to establish the existence of pneumoconiosis through x-ray evidence but was able to prove its existence through medical opinion. Accordingly, in his April 25, 2000 Decision and Order on Remand Denying Benefits, the ALJ did not reconsider the x-ray evidence but focused exclusively on the weight of the medical opinion evidence in concluding that Frasure did not suffer from coal workers' pneumoconiosis. (DX 116).[2] Notwithstanding that the ALJ did not reconsider the x-ray evidence in his most recent decision, I will, in the interest justice, consider the x-ray evidence and the relevant medical opinions to determine whether there was a mistake in determination of fact regarding whether Frasure suffered from coal workers' pneumoconiosis.

Burden of Proof

"Burden of proof," as used in this setting under the Administrative Procedure Act[3] ("APA") is that "[e]xcept as otherwise provided by statute, the proponent of a rule or order has the burden of proof." As used in this context, "burden of proof" means burden of persuasion, not merely burden of production. 5 U.S.C.A. § 556(d).[4] The drafters of the APA used the term "burden of proof" to mean the burden of persuasion. *Director, OWCP, Department of Labor v. Greenwich Collieries* [*Ondecko*], 512 U.S. 267, 114 S.Ct. 2251 (1994).[5]

The claimant bears the burden of establishing the following elements by a preponderance of the evidence: (1) the miner suffers from pneumoconiosis; (2) the pneumoconiosis arose out of coal mine employment; (3) the miner is totally disabled; and (4) the miner's total disability is caused by pneumoconiosis. *Gee v. W.G. Moore and Sons*, 9 B.L.R. 1-4 (1986)(*en banc*); *Baumgartner v. Director*, OWCP, 9 B.L.R. 1-65 (1986)(*en banc*).

A claimant has the general burden of establishing entitlement *and* the initial burden of going forward with the evidence. The obligation is to persuade the trier of fact of the truth of a proposition, not simply the burden of production, the obligation to come forward with the evidence to support a claim.[6] Therefore, the claimant cannot rely on the Director to gather

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[2] As stated previously, the ALJ, therefore, did not the issue of total disability. (DX 116).

[3] 33 U.S.C. § 919(d) ("[N]otwithstanding any other provisions of this chapter, any hearing held under this chapter shall be conducted in accordance with [the APA]"); 5 U.S.C. § 554(c)(2). Longshore and Harbor Workers' Compensation Act ("LHWCA"), 33 U.S.C. §§ 901-950, is incorporated by reference into Part C of the Black Lung Act pursuant to 30 U.S.C. §§ 932(a).

[4] The Tenth and Eleventh Circuits held that the burden of persuasion is greater than the burden of production, *Alabama By-Products Corp. v. Killingsworth*, 733 F.2d 1511, 6 BLR 2-59 (11th Cir. 1984); *Kaiser Steel Corp. v. Director, OWCP* [*Sainz*], 748, F.2d 1426, 7 BLR 2-84 (10th Cir. 1984). These cases arose in the context where an interim presumption was triggered, and the burden of proof shifted from a claimant to an employer/carrier.

[5] Also known as the risk of non-persuasion. *See* 9 J. Wigmore, *Evidence* § 2486 (J. Chadbourn rev. 1981).

[6] *Id*. *See also White v. Director, OWCP*, 6 BLR 1-368 (1983).

evidence.[7] A claimant bears the risk of non-persuasion if the evidence is found insufficient to establish a crucial element. *Oggero v. Director, OWCP*, 7 B.L.R. 1-860 (1985). Evidence which is in equipoise is insufficient to sustain a claimant's burden in this regard. *Director, OWCP v. Greenwich Colleries, et al*., 114 S.Ct. 2251 (1994), *aff'd sub nom*. *Greenwich Colleries v. Director, OWCP*, 990 F.2d 730 (3rd Cir. 1993). Failure to establish any one of these elements will result in a denial of benefits. *Hall v. Director, OWCP*, 2 B.L.R. 1-998 (1980).

Issues Presented

Several issues that had been previously contested by the Employer were withdrawn at the formal hearing and will be listed below as stipulations. The remaining contested issues are as follows: (1) *Pneumoconiosis*: whether the miner has/had pneumoconiosis as defined by the Act and the regulations; (2) *Causal Relationship*: whether the miner's pneumoconiosis arose out of coal mine employment; (3) *Total Disability*: whether the miner is/was totally disabled; (4) *Causation*: whether the miner's death disability or death was due to pneumoconiosis; (5) *Modification*: whether the evidence establishes a change in condition and/or that a mistake was made in the determination of any fact in the prior denial per 20 C.F.R. 725.310.

Stipulations

At the formal hearing, the following stipulations were made: (1) *Timeliness*: that the claim was timely filed; (2) *Miner*: that the person upon whose death or disability the claim is based is a miner; (3) *Post-1969 Employment*: that the miner worked as a miner after December 31, 1969; (4) *Length of Employment*: that the miner worked at least 14 years in or around one or more coal mines; (5) *Dependency*: that the claimant has 1 dependent for purposes of augmentation; (5) *Responsible Operator*: that the named employer is the Responsible Operator; (6) *Insurance*: that the named employer has secured the payment of benefits (Sec. 423); (6) *Subsequent (i.e. Duplicate) Claims*: that the evidence establishes a material change in conditions per 20 C.F.R. 725.309(c), (d). (Tr. 34-35).

New Medical Evidence

To demonstrate a material change in condition, Claimant submitted new evidence, including DX 134, DX 154, and DX 155. Employer submitted DX 162 and EX 1-6. (Tr. 25-26). This evidence is summarized below.

X-Ray Interpretations

1. DX 155 Radiology Report (Rice) 8-29-00

Facility: Central Baptist Hospital
History: Shortness of breath; chest pain

The heart is slightly large but radiographically compensated. The Groshong catheter is well positioned. There is no pneumothorax. There are chronic pulmonary changes. There is no active infiltrate, mass, or effusion.

[7] *Id*.

Impression: (1) Mild cardiomegaly, compensated; (2) Chronic pulmonary changes; (3) Groshong catheter is well positioned. There is no pneumothorax.

2. DX 162 Chest x-ray Re-Read (Poulos B BCR[8]) 8-26-02 (date of re-read)

Film Quality: Grade 3, overexposure, overlying artifact
Name of Facility: Highlands Regional Medical Ctr.
Date of X-Ray: 5-4-01

Re-Reading of Chest x-ray: The heart and great vessels appear within normal limits. The lung fields are clear. Bony thorax and diaphragm shadows appear within normal limits. There is an old fracture of the right 6th rib.

Impression: Negative chest; there is no evidence of pneumoconiosis.

3. DX 162 Chest x-ray Re-Read (Poulos B BCR) 8-26-02 (date of re-read)

Film Quality: Unreadable due to over-exposure
Name of Facility: Highlands Regional Medical Ctr.
Date of X-Ray: 1-5-01

4. DX 162 Chest x-ray Re-Read (Poulos B BCR) 8-26-02 (date of re-read)

Film Quality: Unreadable due to over-exposure
Name of Facility: Central Baptist Hospital
Date of X-Ray: 8-28-00

Relevant Examination, Medical Reports and Depositions

1. DX 134 Deposition Testimony of Dr. Ayesha Sikder 8-2-01

Dr. Sikder's deposition was taken on August 2, 2001. (DX 134). Dr. Sikder is Board-certified in Internal Medicine. (DX 134:5). Pulmonolgy represents approximately 90 percent of Sikder's practice. (DX 134:4). She has completed a full fellowship in pulmonary medicine. (DX 134:5). As part of her practice, she regularly treats coal miers with respiratory problems. (DX 134:6).

Dr. Sikder testified that she first saw Frasure on November 4, 1998. (DX 134:7). At that time, Sikder performed a physical examination and conducted tests. (DX 134:7). She stated that Frasure had symptoms of chronic lung disease at that time. (DX 134:7). She also noted his history of working in coal mines, accumulated surface and underground of fourteen years. (DX 134:7). In addition, she noted his smoking history of 50-pack years, the miner having ceased smoking in 1985. (DX 134:7).

According to Sikder, the physical examination demonstrated that Frasure had very diminished air exchange, which "goes with obstructive airways disease." (DX 134:8). The pulmonary function test showed he had severe obstructive airway disease with concomitant restrictive lung disease. (DX 134:8). By concomitant, Sikder meant that he had "reduced FEV1, which is suggestive of airway disease, but he

[8] The abbreviations "B BCR" are used to designate physician qualifications: "B" for "B-reader" and "BCR" for "Board-certified Radiologist."

also [had] reduced FVC, which is suggestive of restrictive airway disease." (DX 134:9). Specifically, she reported that Frasure's FVC was 1.7 liters, which is 40 percent of predicted, and his FEV1 was 0.75, which is 23 percent predicted. (DX 134:9). Sikder testified that, according to the American Thoracic Society classifications, usually less than 40 percent predicted is severe disease. (DX 134:9). Thus, she opined, that 23 percent of predicted represents severe obstructive disease and is suggestive of end stage lung disease (i.e. lung disease that is so far advanced that there will be no meaningful recovery). (DX 134:9). She added that FVC being 40 percent of predicted also suggests restrictive defect. (DX 134:9). Moreover, Sikder considered the test to be valid in that it was very reproducible (i.e. four trials were done that yielded nearly identical values) and the flow volume group was classic for obstructive airway disease. (DX 134:10). From this visit, she concluded that Frasure had coal workers' pneumoconiosis, COPD hypertension, chronic respiratory failure (which is oxygen dependency), and mild renal insufficiency. (DX 134:11).

After November 4, 1998, Sikder continued to treat the miner for lung disease. (DX 134:11). Initially, she treated him once every two-to-three months. (DX 134:12). Since 1999, she had been seeing him approximately once a month. (DX 134:12). Frasure was on various medications, including Theophylline, Proventil, Albuterol Nebulizer, Atrovent (added to his regimen by Sikder), Albuterol inhaler (as needed), IV Solu-Medrol, and IV Aminophylline (three times a week). (DX 134:12).

At the time of the deposition, Sikder was Frasure's treating physician. (DX 134:12). She reported that since 1998, Frasure had been admitted to the hospital approximately once a month. (DX 134:12). She treated Frasure during all of his hospitalizations. (DX 134:12). Most of his admissions had been due to his respiratory disease, though one or two were for other reasons. (DX 134:12). At her deposition, Sikder testified that Frasure was "severely respiratory symptom" [sic]. (DX 134:13). She noted that Frasure was confined to his bed, oxygen dependent, and steroid dependent such that he had to take oral Prednisone every day. (DX 134:13). She testified that his symptoms were chronic and worsened with minimal exertion (i.e. even turning around in bed caused him to get short of breath). (DX 134:13). He was totally dependent on around-the-clock nebulizer. (DX 34:13). He also used bronchodilators. (DX 134:13). Sikder testified that steroids are given to patients with end stage lung disease for bronchodilation and also, at times, to slow down cachexia (i.e. to maintain a patient's weight). (DX 134:13). Based on his fourteen-year history of underground and surface exposure to coal dust, Sikder opined that Frasure had coal workers' pneumoconiosis. (DX 134:14-15).

In addition to his coal mining history, Sikder based her opinion on Frasure's pulmonary function study and chest x-rays. (DX 134:15). She reiterated her conclusions about the pulmonary function study that she stated earlier. (DX 134:15). She testified that she had looked at Frasure's chest x-rays on several occasions and noticed chronic infiltrates, which are suggestive of coal dust exposure. (DX 134:15). She stated that she had looked at "probably 40 x-rays." (DX 134:16). She also stated that Frasure did have the classic hyper-inflation seen in emphysema. (DX 134:16).

In opining that Frasure's chronic lung disease was due to his coal dust exposure, Sikder did consider his smoking history and admitted that his smoking had contributed to his chronic lung disease. (DX 134:17). However, based on his chest x-ray findings and pulmonary function study, she believed that Frasure "clearly had black lung disease." (DX 134:17). She noted that he also had emphysema, which is caused by both cigarette smoking and coal dust exposure, and that Frasure's disease is due to both causes. (DX 134:17). She stated that Frasure's coal mine dust exposure was a significant cause of Frasure's lung disease. (DX 134:17).

Sikder described chronic obstructive pulmonary disease (COPD) as a disease that most of the time is attributable to cigarette smoke but may also be attributable to coal dust exposure in certain instances. (DX 134:18). She stated that chronic obstructive pulmonary disease described Frasure's chronic dust disease. (DX 134:18). Sikder testified that Frasure has not had the capacity to perform any kind of work activity since 1998. (DX 134:18).

Sikder noted that Frasure also has prostate cancer and that the disease had spread to the bones. (DX 134:18-19). She testified that he also had depression and cor pulmonale. (DX 134:19). She also

mentioned that he had a history of atrial arrythmias (irregular heart rate) due to low oxygen carrying capacity. (DX 134:19). The cor pulmonale was due to right ventricular or right heart failure due to chronic lung disease. (DX 134:19). He had congestive heart failure due to right ventricle failure. (DX 134:19). The basis for Sikder's determination that Frasure had cor pulmonale was the presence of peripheral edema and the absence of any other causes. (DX 134:20). She noted, for example, the absence of coronary artery disease. (DX 134:20).

During her deposition, Sikder referred to a letter dated September 25, 2000 she had written to the BRB in which she noted that Frasure's chest x-rays demonstrate bilateral fibrotic changes and hyperinflated lung fields. (DX 134:21). She elaborated that he had some nodular and some linear changes, which are consistent with coal dust exposure/coal workers' pneumoconiosis. (DX 134:21). One of the x-rays she reviewed had hyperinflated lung fields but overwhelmingly they did not; they just showed chronic infiltrates. (DX 134:21). Sikder opined that the cause of the chronic infiltrate or bilateral fibrotic change was coal dust exposure. (DX 134:21).

During her deposition, Sikder was presented with a summary of x-ray interpretations that were submitted in the case in the previous decisions. (DX 134:21). The summary included readings by B-readers and Board-certified radiologists. (DX 134:22). Sikder was questioned as to whether her weight of the x-ray interpretations was negative for classic medical pneumoconiosis. (DX 134:22). She stated that her opinion would not change because often chest x-rays are negative in a patient with coal dust exposure and this later becomes revealed when lung biopsies are done. (DX 134:22).

During her deposition, Sikder was also presented with a summary of pulmonary function tests that were submitted in the case in the previous decisions. (DX 134:22). Contrary to changing her opinion concerning Frasure, Sikder testified that the summary further substantiated her opinion because most of the FEV1s were about one meter [sic] or less. (DX 134:23).

During her deposition, Sikder was also presented with a summary of arterial blood gas study results that were submitted in the case in the previous decisions. (DX 134:23). She stated that arterial blood gases are in no way correlated with coal workers' pneumoconiosis, and therefore would not change her opinion. (DX 134:23). After examining the oxygen values on the summary, Sikder commented that Frasure had mild hypoxemia (she noted that some of the tests showed moderate hypoxemia). (DX 134:24). She also commented that all of the blood gases were not normal in that they were low. (DX 134:24). She explained that, "unless it goes below a certain very critical value, they do not reflect a person's pulmonary symptomatology because one could be breathing fast enough and maintaining oxygen at a high level." (DX134:25).

2. DX 134 Pulmonary Consultation Summary (Sikder) 11-4-98

Frasure has severe COPD, long standing hypertension, and coal workers' pneumoconiosis. Patient states that his breathing has been worse in the last two years and he has been oxygen dependent since May, 1998. He is an ex-smoker.

Presents with severe dyspnea and a feeling of "can't breathe." He has frequent wheezing and his symptoms are worse at night. He is short of breath with minimal conversation. He however denies any chronic cough. In addition to oxygen he is also on Solu-Medrol home infusion for the past 4-5 months. Patient takes multiple inhalers having best relief with Albuterol MDI. Patient has quit smoking 13 years ago secondary to development of dyspnea. He further admits to dyspnea during meals. He has orthopnea. Denies PND. Denies leg edema.

Past Medical History: Per HPI. Denies history of childhood asthma. Fluvax 1997. Pneumovax 1997.

Past Surgical History: Groshong catheter placement.

Medications: Proventil 4 mg. b.i.d., Albuterol MDI, Theophylline 300 mg. b.i.d., Solu-Medrol and Aminophylline I.V. 3 times/week. Albuterol nebulizers, Sular, and Lasix.

Family History: Significant for asthma. No related medical illnesses.

Allergies: Denied

Social History: Smoker 50 pack years. Quit 13 years ago. Denies ETOH.

Occupational History: Retired 1984. Exposed to 14 years of surface and underground coal mining. Denies any other significant occupational exposure.

Review of Systems: Denies nausea, vomiting, diarrhea, abdominal pain, or change in bowel habits. Admits to urinary hesitancy. Denies any dysuria or increased frequency. Denies any changes in weight. Denies any chest pain, palpitations, dizziness, or diaphoresis. All other systems noncontributory.

Physical Exam: Well developed thin elderly male WM in mild respiratory distress.

Vital Signs:
Pulse 104 BP: 130/80 Resp: 18 Temp: 98.2 Ht: 71″ Wt 173 lbs
O2 SAT: 96% PEFR: 230 cc's

HEENT: Rhinophyma. There is occasional accessory muscle use. No JVD. No adenopathy. Trachea midline. No thyromegaly.

3. DX 134 BRB Letter (Sikder) 9-25-00

4. DX 134 Discharge Summary Dictated (Sikder) 4-13-00

Diagnosed Frasure as having: (1) pseudomonas pneumonia; (2) COPD; (3) coal workers' pneumoconiosis; (4) status post acute respiratory failure; (5) anemia; (6) ischemic colitis; (7) steroid myopathy; (8) SVT; and (9) cachexia. She noted that the patient had been bedridden since December, 1999. He was presented to the hospital with acute respiratory arrest. His blood gases revealed a pH of 7.47, pO2 of 44 on 3 liters of nasal cannula. Patient had to be intubated in the emergency room. His x-rays on admission were clear. However, there was right hilar prominence. The patient was assessed to be in profound sepsis and respiratory failure due to COPD exacerbation. Because of the hilar prominence, a CAT scan of the chest was done which revealed basically a right hilar pneumonia. There was no masses or adenopathy seen. Empirically the patient was started on Claforan on the day of admission with Zithromax IV. He was also started on Solu-Medrol, Digoxin, Theophyllin and bronchodilators.

On 4/9: Sputum culture came back as Pseudomonas. Has Clarofan was discontinued. Patient was started on Levaquin.

On 4/10: Patient was awake breathing comfortably.

On 4/11: Patient was without any new events. He was having significant bronchorrhea. Solu-Medrol was reduced to 40 mg q 8.

On 4/12: Patient had no further dyspnea. He was able to cough up sputum which was no thin and nonpurulent. Solu-Medrol was discontinued. Patient was started on Prednisone 20 mg q d. Levaquin was changed to p.o.

On 4/13: Day of discharge. Patient was without any new symptoms. He remains bedridden and has severe weakness of the lower extremities. However, he has only toe movement and slight lateral rotation.

5. DX 134 Emergency Department Record (Albaree) 4-6-00

Patient is the source of the history. Trouble breathing. Presents with a history of severe shortness of breath at rest. Has underlying COPD. No history of any chest pain. Nothing in the history or presentation to suggest hyperventilation. No history of chronic renal failure. Has a coarse cough. No sputum. Acute exacerbation of chronic bronchitis. Complains of subjective fever ("felt hot") but did not document termperature. History of underlying COPD.

Pulmonary exam: Patient is acutely uncomfortable and in severe respiratory distress. Using accessory muscles to maintain respiratory effort. No evidence of upper airway obstruction or stridor. He has a coarse cough. Coarse rhonchi are heard in the right and left lung fields. Ausculation of the right and left lung fields reveals localized expiratory wheezing.

Cardiovascular exam: Heart tones are irregular and suggestive of atrial fibrillation. Patient has a resting tachycardia.

Musculoskeletal exam: Normal muscle strength and tone is present. NO signs of acute arthritis, clubbing, or significant edema noted.

Interpretation of Tests:

09:40 X-ray 1 view of the chest: These films were reviewed in the clinical context of this case. X-ray shows air trapping consistent with emphysematous changes. There is a localized patchy infiltrate in the right lung field. Based on clinical data and radiographic presentation this is most likely a community acquired pneumonia.

10:01 X-ray 1 view of the chest: These films were reviewed in the clinical context of this case. ET tube is ell [sic] placed above bifurcation or main stem bronchi.

10:02: A baseline atrial fibrillation is noted on cardiac monitor.

Procedure Notes:

9:51: Indication for Intubation: Acute respiratory failure. Received anesthesia prior to intubation. Endotracheal intubation performed with a # [7.5] French ET tube. No complications during procedure.

9:51: Patient sedated. Continuous pulse oximetry monitoring during procedure. Patient tolerated procedure with no complications. Respirator settings: 100% FIO 2. Rate set to 14.

Symptom and Problem List: Acute respiratory distress. Respiratory failure. Retractions. Expiratory wheezing. Non-specific emphysema. Community acquired pneumonia. Acute exacerbation of COPD. Hx of Low grade fever. Tachycardia Atrial Fibrillation. Cough. Old age debility. Abnormal chest x-ray.

Final Diagnosis: 1) Acute respiratory distress; 2) Respiratory failure; 3) Community acquired pneumonia; 4) Acute exacerbation of COPD; 5) Atrial Fibrillation.

Physician Disposition: Condition: Critical. Discussed case with private medical physician including follow up and condition in department.

10:02: Admit to ICU. Discussed case with Dr. [Sikder] who will admit to their service.

6. DX 134 History & Physical Critical Care Note (Sikder) 4-6-00

Patient has complex obstructive airways disease including end-stage chronic obstructive pulmonary disease, advanced coal workers' pneumoconiosis, and chronic respiratory failure.

Patient has been basically steroid-dependent since November 1999 and has been admitted to the hospital several times with chronic obstructive pulmonary disease with exacerbation. Although the patient has steroid myopathy, he is unable to be weaned from the steroids due to his severe respiratory-symptom limitation.

Most recently, the patient was admitted to Highlands Regional Medical Center on 3/2/00 and discharged on 3/17/00. Initially, the patient presented with hyponatremia, dehydration and confusion that was felt to be due to his hypoxia-related because his work up was essentially negative. [sic] The patient was most recently seen a few days ago in the office where he complained of baseline dyspnea with mild bronchorrhea. The patient was being continued on maintenance dose of prednisone and aggressive bronchodilator regimen. He is bedridden.

The patient presented on the day of admission to the emergency room with acute worsening of his dyspnea, cough and purulent sputum since early morning. On the day prior to admission the patient had mild dyspnea that lasted less than ½ hour. There is no report of fever, chills, or any chest pain according to the family. In the emergency room the patient was found acutely dyspnea and gasping for air. His pO2 was 44 with a pH of 7.47 on 3 liters nasal cannula. The patient was, therefore, promptly intubated. When seen at the bedside shortly after intubation, the patient was somewhat combative and anxious but not in any respiratory distress.

Past Medical History: Is per HPI. The patient is oxygen-dependent for the past 2-3 years. In 1998 the patient had been IV steroid-dependent that was successfully wanted towards the end of 1998.

Family History: Is significant for asthma.

Occupational History: History of coal mining. Retired in the 1980s.

Social History: Ex-smoker 50 pack years. Quit 1985.

Physical Examination:

Lungs - Poor exchange bilaterally. Bilateral rhonchi appreciated. Prolonged expiratory phase. There is coarse crackles at the right lung base.

Extremities – No clubbing, cyanosis, or edema.

Laboratory Examination: pH 7.48, pO2 44, pCO2 35 prior to intubation. Chest x-ray shows a right lower lobe pneumonia with prominence of the right hilum.

Impression: (1) Acute respiratory failure secondary to acute pneumonia; (2) Sepsis most probably secondary to pneumonia; (3) chronic obstructive pulmonary disease with exacerbation; (4) pneumonia; (5) coal workers' pneumoconiosis; (6) history of supraventricular tachycardia secondary to hypoxemia; (7) chronic wasting secondary to end-stage chronic obstructive pulmonary disease; (8) steroid myopathy; (9) immobility with chronic bedridden state; (10) osteoporosis; (11) history of chronic polyp; (12) history of GI bleeding.

Discussion: Patient has end-stage chronic obstructive pulmonary disease and end-stage lung disease. Patient present in profound sepsis and respiratory failure secondary to pneumonia. At this time will start the patient on Claforan and Zithromax until cultures available. Will start him on Solu-Medrol 80 mg q 8. Will optimize his bronchodilator therapy. Patient is critically ill.

7. DX 134 Discharge Summary (Sikder) 6-2-00

Final Diagnosis: (1) chronic obstructive pulmonary disease exacerbation; (2) acute bronchitis; (3) coal workers' pneumoconiosis; (4) chronic respiratory failure; (5) steroid myopathy; (6) cachexia; (7) atrial arrhythmia; (8) bedridden state.

Hospital Course: Patient has severe end-stage coal workers' pneumoconiosis and chronic obstructive pulmonary disease. Patient is known for his multiple admissions to the hospital for chronic obstructive pulmonary disease exacerbation. He has a history of intubation in the past two months.

Patient presented because of worsening dyspnea, cough and sputum production. His ABG on 2 liters nasal cannula on admission showed pO2 84, pH 7.40, and pCO2 46. I have noticed that the patient is steroid dependent on Prednisone 10 mg q d. He is also oxygen dependent for the past several years. His chest x-ray on admission did not reveal any acute abnormalities. The laboratory exam was essentially normal.

On 5/31/00, resuming service, the patient appeared to have baseline dyspnea. He was sitting up in bed. It may be pointed out that patient has steroid myopathy and has had physical therapy and difficulty sitting up in bed. While in the hospital, physical therapy was reinstated and the patient was able to stand up on the side of the bed with help.

On 6/1/00, the day of discharge, the patient had baseline cough. His lungs had a few rhonchi which is baseline and he has good air exchange.

8. DX 134 Emergency Department Record (Joudeh) 5-31-00

Trouble breathing. The history begins with a sense of not breathing "normally" with increased effort and shortness of breath. Presents with a history of moderate shortness of breath at rest. There has been a decrease in exercise capacity. With shortness of breath on exertion. Patient becomes short of breath just walking across the room. These symptoms developed rapidly over a period of several hours. Has underlying COPD. No obvious change in treatment to precipitate this event. Nothing in history to suggest orthopnea. No history of PND. Hi history of pedal edema. The patient has noted a decrease in exercise tolerance. Denies fever. No cough or sputum production. No history of hemoptysis. There is a history of audible wheezing. The patient has been using inhalers more than usual.

General History and problem specific ROS: No history of chest pain.
PMH/ROS: No prior history of CHF or significant cardiac risk factors. History of underlying COPD. The patient has been intubated in the past. Had pneumonia in the past. Except as noted the remainder of the Past Medical History and Review of Systems are all negative.

Physical Exam:
General Presentation: The patient appears generally well developed with no significant signs of debility. On exam this patient appears to be acutely ill.
Pulmonary Exam: Patient is in moderately acute respiratory distress but appears well compensated. Intercostal retractions noted. Using accessory muscles to maintain respiratory effort. The respiratory exam is consistent with a true tachypnea. The trachea is midline with no evidence of deviation. Has diffuse auscultory findings. There are decreased breath sounds throughout. Ausculation of reveals generalized expiratory wheezing. [sic]
Musculoskeletal Exam: No significant peripheral edema.

Interpretation of Tests:
X-ray 1 view of chest – these films were reviewed in the clinical context of this case. Non specific pulmonary scaring [sic] and fibrosis noted on chest x-ray.
ABG results: 1-2 liters/min 02. via nasal prongs. On this supplement the pulse oximetry is 83 mm. oxygen. pCO2 45 mm, CO2 ph 7.40.

Symptom and Problem List: Acute respiratory distress. Respiratory distress/tachypnea. Trouble breathing. Retractions. Mild hypoxemia. Acute exacerbation of COPD. Non-specific pulmonary firbrosis. Fever Hx of pneumonia NOS Smoking disorder.

9. DX 134 History & Physical (Caruso) 5-31-00

Chief Complaint: Shortness of air.

History of Present Illness: Patient has extremely severe chronic obstructive pulmonary disease under previous ventilator management in the hospital on previous admission, chronic steroid dependence who comes to the emergency room in the middle of the night complaining of extreme shortness of air and breathlessness at rest. Patient denies chest pain. Patient complains of a slightly increased cough compared to his usual chronic baseline cough, mildly increased sputum production. Patient describes an increased amount of weakness. He cannot walk even short distances. He says that he is too weak to even pick his legs up off the couch when he is lying down.

Medications: Takes Prednisone 20 mg a day every day. He is on multiple inhalers. Cannot remember other medications.

Physical Examination: Lungs have breath sounds bilaterally equal and he has prolongation of expiratory phase with decreased breath sounds in all lung fields. Cannot appreciate wheezes, rales, or other adventitious sounds. Legs are without edema at this time.

Laboratory Data: Blood gas showed pH of 7.4, pO2 on two liters of oxygen supplementation being 84 mmHg, pCO2 of 46 with a base excess of 2.9.

Impression: (1) Chronic obstructive pulmonary disease. Extremely advanced and now with exacerbation. On previous admission the patie-nt required ventilator management; (2) Steroid dependent; (3) Numerous other medications.

10. DX 134 Discharge Summary (Sikder) 9-6-00

Diagnoses: (1) Dissemianted intravascular coagulation; (2) Chronic obstructive pulmonary disease with exacerbation; (3) Acute respiratory insufficiency secondary to chronic obstructive pulmonary disease; (4) coal workers' pneumoconiosis; (5) bronchitis; (6) anemia; (7) severe thrombocytopenia; (8) left flank and chest wall hematoma; (9) osteoporosis; (10) steroid myopathy.

Hospital Course: Patient has severe end-stage chronic obstructive pulmonary disease and steroid myopathy. Patient is steroid and oxygen-dependent. He is known for his multiple admissions for chronic obstructive pulmonary disease with exacerbation. During the course of the year, he has been admitted to this hospital with several chronic obstructive pulmonary disease exacerbations. Since November 1999, he had a bout of severe exacerbation and has been bedridden since. Patient had pain in the back. In the past, his work up had been consistent with chronic obstructive pulmonary disease. He had normal PSA 4 months ago.

Patient presented to the emergency room with worsening dyspnea, cough, and weakness. His chest x-ray is unremarkable. On examination, he was found to have a let flank hematoma which was fairly large (approx. football size).

On 8/25: Dyspnea was slightly better. He had course [sic] breath sounds. Transferred to ICU for monitoring. Patient had an episode of tachycardia. It was felt that hematoma needed to be closely observed.

On 8/26 and 8/27: Patient was started on Levaquin and Zosyn secondary to continued dyspnea, cough, and sputum production. It was felt that sepsis needed to be ruled out.

On 8/28: Hematoma had progressed further. Dyspnea was slightly worse and he had occasional rhonci. Transferred in critical but stable condition to Central Baptist Hospital.

11. DX 134 Emergency Department Record (Styer) No Date

Trouble breathing. The history begins with a sense of not breathing "normally" with increased effort and shortness of breath. Has underlying COPD. No obvious change in treatment to precipitate this event.

Cough. Has a coarse cough. Whitish sputum. Acute bronchitis.

History of underlying COPD

12. DX 154 History and Physical (Stumbo) 10-24-01

Reason for admission: UTI, dehydration, metastatic prostate cancer.

13. DX 154 Consult Sheet (Sikder) 10-24-01

Final diagnosis: Clinical dehydration; UTI

14. DX 154 Emergency Dept. Record (Albaree) 10-24-01

Final diagnosis: Clinical dehydration; UTI

15. DX 154 Discharge Summary (Sikder) 8-24-01

Final Diagnosis: Chronic obstructive pulmonary disease with exacerbation; (2) Chronic respiratory failure; (3) Bronchitis; (4) Coal workers' pneumoconiosis; (5) Altered mental status most probably medication-induced; (6) History of atrial arrhythmia; (7) Hypoxemia related; (8) Steroid myopathy; (9) Cachexia; (10) Anxiety with end-stage lung disease.

Hospital Course: Patient was started on Claforan one gram IV q.8, Solu-Medrol 80 mg q 8 and continued on his home medications including theophylline, albuterol, Serevent, Flovent. The patient is on MS contin at home and Roxanol q. 3 hours p.r.n., which were also continued.

On 8/14, patient complained of pain in the bones. Roxanol was continued. He had poor air exchange with rhonchi; albuterol nebulizers were increased to q.3 hours p.r.n.

On 8/15, patient complained of persistent back pain and constipation. Sorbitol was given. MS contin was increased to 45 mg b.i.d. X-ray of the spine was done, which showed osteoporosis of the spine with old compression fractures. He was also started on Pulmocare.

On 8/16, back pain was improved. Dyspnea was somewhat improved. His lungs had somewhat increased air exchange. The Solu-Medrol was reduced to 14 mg q. 8 hours.

Patient's lab exam revealed anemia with hemoglobin of 11 to 12 and mild leukocytosis. Had some left shift on differential. Initial chemistries revealed glucose of 132, therefore, his glucose was better controlled. His alkaline phosphates was 1544. It was felt to be due to bone metastasis.

On 8/17, his dypsnea was better.

On 8/18, patient complained of increasing weakness.

On 8/19, patient was confused on and off. Roxanol was discontinued due to the confusion and he was started on Percocet and was started on Ambien. Theophylline level was obtained, which was therapeutic.

On 8/20, patient's confusion was unchanged. MS contin was increased to 60 mg q. 12. Due to persistent pain, patient's lung had poor air exchange.

On 8/21, labs showed glucose of 186 at rest with mild hyponatremia and hypokalemia. Long discussion was carried out that patient's overall condition, COPD, steroid myopathy, etc. was worse. CT of head was obtained to rule out brain met and CT of the head was unremarkable.

On 8/22, patient had metabolic encephalopathy most probably medication induced. Ativan was given of agitation. Patient started on multivitamin.

On 8/23, air exchange improved.

On 8/24, COPD was felt to be baseline. Patient discharged home with following medications: MS contin 30 mg b.i.d., Percocet one tablet q. 4 hours for breakthrough pain. He was given 50 pills. MVI one tablet q. day, Ativan 0.5 mg q.h.s. p.r.n., Ceclor 500 q. 12 three days. Advised to resume other home medications.

16. DX 154 History & Physical (Sikder) 8-15-01

Chief complaint: Chronic obstructive pulmonary disease exacerbation

History of Present Illness: Known to Highlands Regional Medical Center for Chronic obstructive pulmonary disease exacerbation. Has underlying coal workers' pneumoconiosis and chronic respiratory failure. Is bedridden secondary to all of the above. Also has steroid myopathy and cachexia of end-stage lung disease. Also has carcinoma of the prostate with bone metastases.

Patient has baseline severe respiratory symptomatology who needs help turning in bed and is unable to ambulate. He has chronic dyspnea around the clock. MDI use q.3h and baseline wheezing. His dyspnea was worse for 2-3 days. His respiratory symptomatology has progressed over the past 2-3 days and his dyspnea was particularly worse on the day of admission. He was given Depo-Medrol and Xopenex nebulizer treatment in the office without any improvement. Patient denies any fever or chills. Admits to cough. Difficulty raising sputum. Has chronic low back pain which is worse. Patient has baseline pedal edema. Denies any worsening. Further denied any pleuritic chest pain.

Past Medical History: Related in HPI. On estrogen replacement for his carcinoma of prostate. Patient had DIC in 2000 because of disseminated carcinomatosis, pathological fracture of the hip also in 2000, history of Pseudomonas, pneumonia, severe osteoporosis secondary to chronic Prednisone, chronic steroids therapy, atrial arrythmias, chronic dyspnea, depression, vertebral compression fracture, chronic constipation secondary to narcotics and cor pulmonale.

Social History: Tobacco 50 pack years. Quit in 1985. Denies ETOH.

Occupational History: Coal mining, surface and underground. Retired in 1980. Currently disabled. Bedridden.

Physical Examination:
Lungs; scattered rhonchi bilaterally. No rales appreciated. Expiratory phase prolonged. Air exchange is poor.
Extremities: No clubbing or edema

Impression: Chronic obstructive pulmonary disease with exacerbation; bronchitis; cor pulmonale; coal workers' pneumoconiosis; chronic respiratory failure; carcinoma of prostate with bone mets; history of atrial arrhythmias; steroid myopathy; cachexia; anxiety of end-stage lung disease

17. DX 154 Discharge Summary (Sikder) 6-11-01

Final Diagnosis: 1) Chronic obstructive pulmonary disease exacerbation; 2) Bronchitis; 3) Coal workers' pneumoconiosis; 4) chronic respiratory failure; 5) cachexia; 6) carcinoma of the prostate; 7) bone metastases; 8) steroid myopathy; 9) chronic constipation; 10) degenerative joint disease; 11) gastroesophageal reflux disease

Patient has end stage chronic obstructive pulmonary disease currently bedridden and steroid myopathy. Patient is Prednisone dependent and oxygen dependent. Presented with acute dyspnea which was refractory to outpatient therapy. Patient was started on Levaquin and IV Solu-Medrol and admitted to hospital. Patient had an episode of arrhythmia and his last admission was felt to be hypoxia related. Due to chronic lung disease, patient was on Digoxin for his atrial arrythmia. On admission his ABG showed pH of 7.31, pCO2 of 54, pO2 of 83 on two liters nasal cannula. His chest x-ray was unremarkable. On 6/10, patient still had wheezing. He was continued on his current medications. On 6/11, day of discharge, patient had baseline dyspnea. There was no wheezing. His lungs were clear.

18. DX 154 History & Physical (Sikder) 6-6-01

Reason for Admission: Chronic obstructive pulmonary disease exacerbation

Patient has severe advanced chronic obstructive pulmonary disease and coal workers' pneumoconiosis. Also has multiple other medical problems including steroid myopathy, cachexia, and chronic respiratory failure due to his longstanding chronic obstructive pulmonary disease. Currently confined to the bed. Most recently admitted to the hospital on 5/4/01 with chronic obstructive pulmonary disease exacerbation. Chest x-ray at that time did not reveal any infiltrate in the lung. Patient remains Prednisone and oxygen dependent for the past year. Has been oxygen dependent for several years but has been Prednisone dependent for more than a year.

Presented with increasing dyspnea. Patient's dyspnea has been gradually progressing over the past two weeks. He admits to cough which is nonproductive but he has difficulty raising sputum and has had low grade temperatures. Patient has had weakness which prevented him from continuing with physical therapy. When seen in the office he had audible wheezing and was in mild respiratory distress.

Past Medical History: Has carcinoma of the prostate with bone mets, osteoporosis, degenerative joint disease, and has got a compresison fracture of the T spine and LS spine. Status post hip fracture. Patient has cor pulmonale and LV dysfunction, failure to thrive, chronic dyspepsia, bedridden state, and chronic constipation.

Family History: Significant for asthma.

Social History: Tobacco 50 plus pack years. Patient quit in 1985. Denies ETOH.

Occupational History: Coal dust exposure for approximately 30 years. Patient retired in 1980.

ROS: Occasional pedal edema.

Physical Examination:
Lungs: Poor air exchange bilaterally with audible and ausculatory rhonchi. Expiratory phase is significantly prolonged.
Extremities: No clubbing or edema.

Impression: 1) Chronic obstructive pulmonary disease exacerbation; 2) Coal workers' pneumoconiosis; 3) Chronic respiratory insufficiency; 4) Steroid myopathy; 5) Severe degenerative joint disease; 6) Carcinoma of the prostate; 7) Bone mets; 8) Compression fractures; 9) Osteoporosis

19. DX 154 Discharge Summary (Sikder) 5-24-01

Final Diagnosis: 1) Chronic obstructive pulmonary disease exacerbation; 2) Bronchitis; 3) Coal workers' pneumoconiosis; 4) Cor pulmonale; 5) Osteoporosis secondary to chronic steroid therapy; 6) Steroid myopathy; 7) Carcinoma of the prostate with diffuse skeletal mets; 8) Cachexia; 9) Bedridden state

Patient has severe coal workers' pneumoconiosis/COPD. Patient presented with exacerbation to the office. Patient's chest x-ray was without any acute infiltrate; however, there was evidence of skeletal mets.

20. DX 154 History & Physical (Sikder) 5-4-01

Reason for Admission: COPD exacerbation

Clinical Case Summary: Patient has severe end-stage COPD, coal workers' pneumoconiosis, chronic respiratory failure and is currently bed confined. Ambulates very limited with a wheelchair. Patient has been several times to the hospital with COPD exacerbation. Has CA of the prostate with bone mets. He is status post pathological fracture of the left hip.

Presented to the office with worsening of his baseline cough and sputum production. Patient is known to have an audible wheeze. At home, patient is maintained on Theophylline, Albuterol nebulizer, and Combivent with persistence of symptoms. He has chronic baseline dyspnea and wheezing. For past two days, patient has been having worsening of his baseline symptoms with difficulty raising sputum. Patient's dyspnea is worse during meals forcing him to reduce his p.o. intake. Denied any fever or chills.

Occupational History: Significant for asthma

Social History: Ex-smoker 50-pack years. Quit in 1985. Denies ETOH.

Physical Examination:
Lungs: Poor air exchange with significantly prolonged expiratory phase. Has bilateral inspiratory and expiratory rhonchi.

Extremities: No clubbing. No edema.

Impression: 1) COPD exacerbation; 2) Bronchitis. Rule out pneumonia; 3) Coal workers' pneumoconiosis; 4) Cachexia; 5) CA of prostate with bone mets; 6) Osteoporosis; 7) Cor pulmonale; 8) Chronic respiratory failure; 9) Steroid myopathy; 10) Compression vertebral fracture old; 11) Gynecomastia secondary to hormone replacement therapy for CA of prostate; 12) Chronic constipation; 14) Anxiety; 14) Depression

The patient will be started on IV Clarofan. Biaxin and IV Solu-Medrol.

21. DX 154 History & Physical (Stumbo) 9-6-00

Chief Complaint: Chronic obstructive pulmonary disease with exacerbation

History of Present Illness: Long history of end-stage chronic obstructive pulmonary disease with multiple admissions here. Poor historian – has no clue as to what medications he's on. Reports complaining of chronic shortness of breath although increased over the last one day. Reports some cough but no sputum production. No chest pain. Complains of pain and bruising in his left flank. Reports low grade fever, some chills but again denies frank chest pain or productive cough.

Social History: Patient has a long history of tobacco abuse but none in 15 years.

Family History: Noncontributory

Physical Examination: The patient is a very frail cachectic white male in moderate respiratory distress.

On 8/25 hematoma appeared slightly smaller. His dyspnea was slightly better. He had course [sic] breath sounds. Patient was transferred to Intensive Care Unit for monitoring. Serial CBCs were ordered. The patient had an episode of tachycardia. He was started on a Cardizem drip.

On 8/26 and 8/27 patient was started on Levaquin and Zosyn secondary to continued dyspnea, cough, and sputum production. It was felt that sepsis needed to be ruled out.

On 8/28, patient had multiple ecchymosis rather large and generalized and his hematoma had progressed further. Patient's dyspnea was slightly worse and he had occasional rhonchi. It was felt that patient was in DIC and that he needed to be monitored in a center where hematology services were available 24 hours and that carcinomatosis needed to be ruled out. PSA was ordered. However, it was not performed secondary to the patient being transferred. He was transferred in critical but stable condition to Central Baptist Hospital.

22. DX 154 Discharge Summary (Sikder) 1-26-01

This visit was related to a hip fracture.

23. DX 154 History & Physical (Sikder) 1-4-01

This visit was related to a fall that resulted in a hip fracture.

24. DX 154 Discharge Summary (Sikder) 3-9-01

Regarding patient's fall and hip fracture.

25. DX 154 Emergency Dept. Record (Albaree) 1-4-01

Regarding patient's fall and hip fracture.

26. DX 154 Discharge Summary (Sikder) 2-24-00

Patient has chronic respiratory failure, severe end-stage chronic obstructive pulmonary disease, coal workers' pneumoconiosis, who has been having wasting syndrome of end-stage chronic obstructive pulmonary disease, severe depression due to end-stage chronic obstructive pulmonary disease and slowly recovering acute bronchitis.

Patient was initially admitted to the acute care setting and transferred to Support Care Unit for rehab and further management.

He was transferred to Support Care Unit. Of note is that patient developed supraventricular arrhythmia due to hypoxia during his stay in the hospital, which was controlled with digoxin.

On 2/21, patient had significant weakness. Was able to stand for only a few seconds. Patient was afebrile. His heart rate remained normal sinus rhythm. Patient was started on supplemental Pulmocare.

On 2/22, patient continued to have generalized weakness and was unable to cooperate with physical therapy. Pulmocare was increased to 1 tablet t.i.d. and normal saline was started. At that time, the diagnosis of steroid myopathy was entertained given that patient had global weakness, which is why Solu-Medrol was discontinued. The patient was started on Prednisone.

On 2/24, it was decided that patient would be better off at home.

On 2/25, patient was discharged home. He remains severely symptom-limited and contained to bed with baseline dyspnea, cough and sputum production. He appetite remained poor.

27. DX 154 History & Physical (Sikder) 2-7-00

Patient has severe end-sage chronic obstructive pulmonary disease, coal workers' pneumoconiosis. Currently oxygen and steroid dependent. Since early January 2000, patient has been on home infusion with IV Sol-Medrolm IV Rocephon, and IVaminophylline initially at three times a week and thereafter he was treated on five times a week. The patient had Groshong catherer placed for the above purposes. Patient's dyspnea, exercise intolerance, cough and wheezing was baseline until 11/99 when his symptoms were progressively worse. Patient has been admitted to the hospital since then approximately four times.

He was most recently seen in the office on 1/28/00 when he complained of persistent wheezing, cough, and sputum production. At that time his home infusions were increased to five times a week and he was given treatment of Zopenex 2.5 mg with some improvement.

Patient continued to have worsening dyspnea and severe chest tightness on the day of admission. His symptoms were progressively worse prompting him to present to the hospital. Denies any fever or chills. Has difficulty expectorating his sputum and denies any chest pain.

Social History: Exsmoker 50 pack years. Quit in 1985. Denies ETOH.

Occupational History: Accumulative coal mining 15 years retired in 1980s. Denies any other industrial exposure.

Family History: Significant for asthma.

Physical Examination:

Lungs: Poor air exchange bilaterally. Bilateral inspiratory and expiratory rhonchi. No crackles at bases.

Extremities: No clubbing or edema.

Impression: (1) Chronic obstructive pulmonary disease with continued exacerbation; (2) acute on chronic respiratory insufficiency; (3) coal workers' pneumoconiosis; (4) cor pulmonale; (5) hypertension.

Discussion: Patient has advanced airway obstruction from a combination of chronic obstructive pulmonary disease and coal workers' pneumoconiosis. Patient is symptomatic in spite of aggressive treatment with bronchodilators in both infusion and oral treatment. Since the symptoms have progressed in spite of the above therapy, the patient needs to be admitted to the hospital. If the patient fails to improve will have to discuss on long term placement therapy.

28. DX 154 Discharge Summary (Sikder) 4-3-00

Final diagnosis: (1) Chronic obstructive pulmonary disease exacerbation; (2) Acute bronchitis; (3) Coal workers' pneumoconiosis; (4) Hyponatremia; (5) Dehydration; (6) Chronic respiratory failure; (7) Osteoporosis; (8) Immobility; (9) Colonic polyps

Hospital Course: Patient has severe end-stage chronic obstructive pulmonary disease/coal workers' pneumoconiosis/chronic respiratory failure. Patient is steroid dependent and oxygen dependent and is known to the hospital for his multiple and frequent admissions. Patient was most recently discharged a week prior to admission after a prolonged stay in the hospital and in the Support Care Unit. He continues to have severe baseline dyspnea, severe exercise intolerance, and episodic wheezing. Patient had been having very poor p.o. intake at home and at the time of admission was found to have hyponatremia with a serum sodium of 123, chloride of 88. Patient has failure to thrive secondary to his severe respiratory symptom limitations and also has constipation. He was started on IV fluids and a regular diet. Patient had worsening baseline dyspnea, cough, and sputum production. Empirically, he was last started on Solu-Medrol 60 mg q. 8 and Claforan 1 gram q.8. His home medications were continued. Of note is that in his last admission, he had supraventricular tachycardia felt to be related to hypoxia which was controlled with digoxin. Pa

On 3/3, patient's serum sodium had improved to 127, however, his dypsnea and cough continued.

On 3/4, patient was started on Pulmocare. Nebulizer treatment was changed to Xopronex. Pulmonary toileting was continued.

On 3/5, dixogin levels were therapeutic, however, the patient continued to have severe respiratory symptom limitation. He was started on Cipro 500 b.i.d. secondary to Pseudomonas in the sputum. Clarofan was discontinued. Solu-Medrol was reduced to 40 mg. q.8.

On 3/6, dyspnea was slightly better. Sputum was less purulent. Pulmocare was increased to q.i.d. and Solu-Medrol was reduced to 40 mg q.12.

On 3/7, patient continued to have copious sputum. He had coarse breath sounds bilaterally. His repeat chest x-ray showed infiltrate. LS spine x-ray showed osteoporosis with compression fracture of T12 which was felt to be old. Chest x-ray, however, showed no infiltrate. In view of the fact that patient was having copious sputum production, bronchospasm, etc., it was felt that the patient should be treated for Pseudomonas pneumonia.

On 3/8, patient was attempted to be mobilized. He was unable to sit up. Patient had severe pain in the lower back when movements were attempted and physical therapy decision was therefore aborted. He continued to have thick purulent sputum. His lungs had a few scattered rhonchi.

On 3/9, the dyspnea was worse. The Solu-Medrol was increased to 60 mg IV q.8. The patient was started on Miacalcin nasal spray secondary to severe osteoporosis felt to be secondary to frequent IV steroid use. Neurology consult was obtained secondary to generalized weakness. At this point it may be pointed out that the patient had been on frequent IV steroids since November of 1999 secondary to continued chronic obstructive pulmonary disease exacerbation. Given his global weakness, lack of focal findings, and normal CT it was felt that the patient had steroid myopathy. His respiratory failure at this point was felt to be acute on chronic. Foley catheter was discontinued but the patient continued to have difficulty urinating. Foley was reinserted. Neurology consult was obtained. An extensive neurological exam was done and it was found that patient had steroid myopathy, however, an EEG was ordered to rule out polymyositis. CPK and ESR were negative again making steroid myopathy possible.

On 3/11 and 3/12, patient continued to have shakiness, dyspnea, and cough and sputum production. His IV medications were continued.

On 3/13, patient's lung was somewhat clearer and he felt somewhat better. Patient's Solu-Cortef was discontinued and the patient was started on prednisone.

On 3/15, patient had diffuse tenderness in abdomen with reduced air exchange.

On 3/17, patient remained dyspneic with movement and was still having poor p.o. intake. Patient sent home.

29. DX 154 History & Physical (Kendrick) 3-2-00

Chief Complaint: Weakness and confusion

History of Present Illness: Patient brought to emergency room with increasing weakness and lethargy and hallucinations over past two to three days. Since his discharge, patient had eaten poorly and complained of increased shortness of breath and cough with sputum production. Denied any fever or chest pain. Has no fevers or chills. Denies any palpitations, syncopal or presyncopal events.

Past Medical History: (1) End-stage chronic obstructive pulmonary disease; (2) Supraventricular tachycardia; (3) Prostate cancer; (4) Coal workers' pneumoconiosis; (5) Hypertension; (6) Cor pulmonale.

Social History: Smoked greater than 50 pack years. Quit in 1985. No use of alcohol.

Family History: Positive for asthma

Physical Exam:
Lungs: Has got some scattered rhonchi with a few expiratory wheezes and decreased air exchange.
Cardiovascular: Tachycardiac
Extremities: Without cyanosis, clubbing or edema

Laboratory Studies: Chest x-ray is reportedly clear. ABG shows a pH of 7.5, pO2 71, pCO2 30 and this is on two liters

Impression & Plan: Chronic obstructive pulmonary disease end-stage. Will empirically place the patient on Claforan and Solu-Medrol and continue him on his nebulization treatments but suspect that he is currently baseline.

30. DX 154 Consultation Report (Sikder requesting; Rao consulting) 3-9-00

Chief Complaint: Acute exacerbation of chronic obstructive pulmonary disease. Patient is referred for neurological assessment of severe motor weakness affecting all extremities.

History of Present Illness: History of chronic hyperstension, chronic obstructive pulmonary disease, prostate carcinomam and supraventricular tachycardia. Patient was recently admitted for worsening of his respiratory status. According to patient's wife, his chronic obstructive pulmonary disease has worsened recently. Since approx 2-4 weeks, he developed worsening of his respirations with bilateral congestion. Patient was admitted at Highlands Regional Medical Ctr. Patient was treated with steroids from 2/18 to 2/25. He was discharged home but again was readmitted with worsening of his respirations. He was noticed to have generalized motor weakness involving both upper and lower extremities since approx 4 weeks. Patient's wife recalls him to have these symptoms more or less within 24-48 hrs. His respirations got worse and he was unable to walk. Since then, the patient cannot get out of bed or lift his arms above the shoulder line. His symptoms have worsened in the past few weeks. There is no associated sensory symptoms. He denies numbness or loss of sensations in lower extremities. No associated bladder incontinence. No history of any saddle anesthesia. Patient complains of mild back pain but otherwise nothing acute. There is no history of trauma to the spine.
Past Medical History: Significant for several medical problems including end stage chronic obstructive pulmonary disease, prostatic cancer, coal workers' pneumoconiosis, hypertension, cor pulmonale. There is no history of stroke or trauma to the spine in the past.

Personal History: Patient has a history of smoking in the past. He is a disabled coal miner.

Physical Exam:
Lungs: The patient has evidence of severe chronic obstructive pulmonary disease with bilateral rales and wheezes.
Cardiovascular: Essentially normal other than tachycardia.
Extremities: There is no pedal edema detected.
Neurological: No evidence of confusion is detected at this time. No evidence of hallucination or delusion noticed.

Clinical Impression: Severe chronic obstructive pulmonary disease and has received large doses of steroids in the past. Patient still needs Solu-Medrol for his respiratory failure. The predominate finding on motor weakness involving both upper and lower extremities symmetrically without sensory loss and absent deep tendon reflexes consistent with myopathy, probably due to steroid-induced myopathy. No evidence of polymyositis is detected. Also, it is unlikely to be inclusion body myositis but it cannot be ruled out.

Plan: Will check CPK and sed rate. Recommend an electromyographic study – can be done as an outpatient. Will start physical therapy. If possible, will decrease his Solu-Medrol and steroids.

31. DX 154 Consultation Report (Sikder requesting; Bahram consulting) 3-14-00

Reason for Consult: Fecal incontinence

32. DX 154 Emergency Dept. Record (Styer) No Date

CC: Shortness of breath. Onset of presenting problem began 1 day ago.

Problem #1: Trouble breathing. History begins with a sense of not breathing 'normally' with increased effort and shortness of breath. Has underlying COPD. No obvious change in treatment to precipitate this event. No fever, significant sputum or other systemic manifestation of infection. No upper airway symptoms, hoarseness, trouble swallowing or loss of voice. Nothing is history to strongly suggest CHF. No orthopnea or history suggestive of PND. No history of any chest pain. Nothing in history or presentation to suggest hyperventilation.

Problem #2: Cough. He has a coarse cough. Whitish sputum. Acute bronchitis.

PMH/ROS: History of underlying COPD. Except as noted the remainder of the Past Medical History and Review of Systems are all negative.

Physical Exam:
Pulmonary Exam: Patient is in moderately acute respiratory distress but appears well compensated. Intercostal retractions noted. Using accessory muscles to maintain respiratory effort. The respiratory exam is consistent with a true tachypnea. The trachea is midline with no evidence of deviation. No evidence of upper airway obstruction or stridor. Has diffuse ausculatory findings. Generally good air exchange. Few crackles heard in all lung fields. Patient has a few scattered wheezes. No significant local ausculatory findings.

Interpretation of Tests: ABG Results: pO2: 69 mm Oxygen on Room Air. pCO2 42 mm CO2 pH 7.41. Arterial blood gas oxygen saturation is consistent with mild hypoxia. The arterial blood pH is within normal limits. Arterial pCO2 is normal.
X-ray view of the chest – these films were reviewed by me in the clinical context of this case. No acute abnormalities seen on chest x-ray. Normal cardiac shadow. No evidence of cardiomegaly. Lung fields are clear. No loss of volume. Has a wide mediastunum on film. No evidence of pleural effusion.

Symptoms and Problem List: Acute respiratory distress. Respiratory distress/tachypnea Retractions Moderate depression of pO2 Miled arterial hypoxia Tachypnea Acute exacerbation of COPD Sputum production Acute bronchitis COPD Hx of COPD Diaphoresis Pallor Anemia Etiology? Cough Old age debility

Final Diagnosis: 1) COPD – acute exacerbation; 2) Anemia – Etiology?

Physician Disposition: Condition – serious. Admit to regular bed.

33. DX 154 Emergency Dept. Record (Albaree) 3-2-00

CC: Shortness of breath. Onset of presenting problem began 1 week ago.

Problems: Shortness of breath. Coarse cough (junky productive cough; acute exacerbation of chronic bronchitis). Trouble breathing (describes a tight chest pain – has an uncomfortable feeling of chest pressure). Hallucinating (seeing things that were not there, talking out of his head).

General History of Problem specific ROS: History of pneumonia.

PMH/ROS: History of underlying COPD. Pos. & Neg.

Physical Exam:
General Presentation: Patient appears to be in moderate distress and is somewhat uncomfortable during the exam. Patient appears to be acutely ill. Skin is pale. Has dry mucous membranes and appears dry. Alert and appropriate during exam. Patient is cachectic and chronically ill appearing. The patient is quite elderly and frail. Patient appears to be generally debilitated.

Pulmonary exam: Patient is in no acute respiratory distress at rest. Has a coarse cough. These symptoms are associated with recurrent episodes of coughing. Acute exacerbation of chronic bronchitis. Coarse rhonchi are heard in the right and left lung fields.
Cardiovascular exam: Heart tones are irregular and suggestive of atrial fibrillation. Patient has a resting tachycardia.
Neurologic exam: Patient is alert and oriented with respect to person, place, and time.

Symptom and Problem List: Chest pain. Significant hyponatremia. Acute and chronic bronchitis. Clinical dehydration. Cachexia. Tachycardia. Tachypnea. Mild hypoxemia. Cough Pallor Old age debility Generalized debility Systolic hypotension (this visit)

Final Diagnosis: 1) Significant hyponatremia; 2) Acute and chronic bronchitis; 3) COPD; 4) Cachexia

Physician Disposition: Condition: Serious. Admit to telemetry bed.

34. DX 154 Emergency Dept. Record (Bagri) 2-2-00

CC: Shortness of breath

Problem #1: Trouble breathing: presents with a moderate shortness of breath at rest. There has been a decrease in exercise capacity. With shortness of breath on exertion. These symptoms developed rapidly over a period of several hours. Has underlying COPD. No fever, significant sputum or other systemic manifestations of infection. Has noted pedal edema. No history of any chest pain. Nonsmoker with no known hypertension or diabetes. Patient is on home oxygen which he uses intermittently.

PMH/ROS: No history of prior heart surgery. Patient is generally well. No history of unexplained weight loss, constitutional symptoms, chest pain, or significant respiratory/cardiac symptoms. The rest of the ROS is negative. Has emphysema.

Family History: Asthma runs in the family; which affects mother.

Social History: Patient is an ex-smoker. Patient is currently receiving home health care. Patient is also currently getting home health care from a visiting RN.

Physical Exam:

Pulmonary Exam: Patient appears to be in mild respiratory distress. Respiratory exam is consistent with a true tachypnea. Patient is on room air. A dry nonproductive cough is noted. Associates with occasional coughing. No sputum. There are decreased breath sounds over the right and left lung fields. No rales or rhonchi heard in the right and left lung fields. Ausculation of the right and left lung fields reveals localized expiratory wheezing.

Cardiovascular Exam: Heart tones are regular. Patient has a resting tachycardia. Normal heart sounds. No audible murmur or rub. No audible gallop.

Interpretation of Tests:

ABG Results: pO2 68 mm Oxygen on Room Air pCO2 43 mm CO2 pH 7.385
X-Ray 1 view of the chest – these films were reviewed by me in the clinical context of this case. No acute abnormalities seen on chest x-ray. No evidence of atelectasis.

Symptom and Problem List: Tachypnea Respiratory distress/tachypnea Moderate hypoxemia Tachycardia Acute exacerbation of COPD Cough Pedal edema Pallor Smoking disorder Family history of asthma

Final Diagnosis: COPD – Acute exacerbation

Physician Disposition: Condition: Stable. Admit to regular bed.

35. DX 154 Discharge Summary (Sikder) 2-23-00

Final Diagnosis: (1) Klebsiella pneumonia; (2) Chronic obstructive pulmonary disease with continued exacerbation; (3) Severe coal workers' pneumoconiosis; (4) supraventricular tachycardia; (5) acute on chronic respiratory insufficiency; (6) hypertension; (7) cor pulmonale; (8) weight loss; (9) steroid myopathy.

Hospital Course: Patient has severe end stage chronic obstructive pulmonary disease, coal workers' pneumoconiosis. Patient has had a progressive downhill course since November of 1999 and has been admitted to the hospital multiple times since then. On this particular admission prior to admission, patient was being treated with Solu-Medrol and IV Rocephin and IV Aminophylline via Groshong catheter and patient continued to get progressively worse. He presented to the emergency room on 2/2/00 with worsening of the baseline dyspnea, continued weakness, and cough and sputum production. Patient was admitted to the hospital on empiric IV Unasyn and Solu-Medrol and continued on his Lasix, Theo-Dur, Atrovent, Ventolin. Patient was also started on aggressive bronchodilator regimen.

On 2/3 because of continued dyspnea his Brethine 2.5 mg t.i.d. was added to his regimen. On admission his Theophylline level was 3.2. His ABG revealed pH 7.38, pCO2 43, pCO2 68. Patient remains severely symptom limited. Chest x-ray was unremarkable.

On 2/4, patient stayed up all night with dyspnea. He was unable to rest. He was started on Restoril same day. Continued to have accessory muscle use, poor air exchange, and diffuse expiratory and inspiratory rhonchi.

On 2/5 and 2/6, patient continued to have bilateral inspiratory and expiratory wheezes with severe symptom limitation. His IV antibiotics and IV steroids were continued. On 2/6 Biaxin 500 b.i.d. was added to his regimen.

On 2/7, patient remained dyspneic which was worse with minimal movement. He was unable to walk to bathroom. Incentive spirometry was started. Patient's Groshong catheter had some oozing. However, it was easy to flush. The Solu-Medrol was reduced to 60 IV q8. Low potassium was replaced.

On 2/8, dyspnea was slightly better. Biaxin was discontinued. Patient, however, continued to have rhonchi which was slightly reduced.

On 2/9, his dyspnea was worse. Patient remained bed ridden. He was given TED stockings. Solu-Medrol was increased to 80 mg q8. Unasyn was continued given the patient had failed outpatient Rocephin and prior to that Clarofan therapy.

On 2/10 patient complained of heartburn. Zantac was added to his regimen. Dyspnea was reportedly slightly improved. Solu-Medrol was again reduced to 60 mg q8.

On 2/11, sputum came back as Klebsiella pneumonia sensitive to Unasym which was continued. Patient was having severe end stage cephalopelvic disproportion maximized on therapy. ABGs done on day of

admission revealed pH 7.47, pO2 58, pCO2 39 on two liters. His F102 was increased to 4 liters and Solu-Medrol was increased to 80 mg q8.

On 2/12 patient was rather dyspneic.

On 2/13 patient developed paroxysmal supraventricular tachycardia at a rate of 150 to 170 which was felt to be hypoxia related. Patient was given IV Calan and Mag. Sulfate and Morphine with low dose Furosemide and was transferred to ICU. Patient was started on and responded to Cardizem drip that night. Heart rate remained 110s. Patient's blood pressure initially unstable thereafter. It was maintained. Brethine and Robaxin were discontinued because of the supraventricular tachycardia.

On 2/14 patient's dyspnea was slightly better. Denied any palpitations. Continued to have accessory muscle use and occasional rhonchi. The 2-D echo was obtained the same day which was essentially normal wall motion. Repeat chest x-ray unremarkable. Telemetry was normal sinus rhythm.

On 2/15 Bactrim DS was added to his regimen. The patient continued to have poor air exchange.

On 2/16 patient was transferred to telemetry. Dyspnea was slightly improved. Solu-Medrol was reduced to 20 mg q8.

On 2/17 patient was started on physical therapy. Had some passive range of motion. However, was only able to raise himself up in bed. Unable to walk to bathroom. Patient preferred to have Foley catheter. Respiratory status was felt to be chronic at this standpoint. Patient was able to cough up his own secretions and air exchange was fair. Transferred to Support Care Unit same day on Rocephon 1 gram q.d. Solu-Medrol 40 mg q.d. and he was continued on Cardizem, Bactrim for seven days, Digoxin, Restoril, and Zantac. Patient was transferred to Support Care Unit in stable but with advanced chronic obstructive pulmonary disease, steroid myopathy, etc.

36. DX 154 Discharge Summary (Sikder) 1-3-00

Final Diagnosis: (1) Chronic obstructive pulmonary disease with exacerbation; (2) Acute on chronic respiratory failure; (3) Bronchitis; (4) Coal workers' pneumoconiosis; (5) Cor pulmonale.
Hospital Course: Patient has severe coal workers' pneumoconiosis and advanced chronic obstructive pulmonary disease. Patient is oxygen-dependent. Has been steroid-dependent in the past but was tapered off approximately a year ago. Patient was treated as an outpatient in November with chronic obstructive pulmonary disease with exacerbation and twice in December with chronic obstructive pulmonary disease with exacerbation. The latter two times he has refused hospitalization. He presented with worsening of his baseline dyspnea. Patient's saturation was worse than baseline. Thus, he was admitted to hospital. His labs revealed a glucose of 164 with the rest within normal limits. His glyocohemoglobin was less than 6. CBC was 15,000 on admission (note the patient has been on steroids a week prior with left shift). Sputum grew pseudomonas. Chest x-ray showed evidence of fibrotic scarring without any acute infiltrates. Electrocardiogram was normal sinus rhythm with chronic obstructive pulmonary disease changes. His arterial blood gas revealed a pH of 7.41, pCO2 37, pO2 79 on room air. Empirically, he was started on Solu-Medrol 80 mg q.8 and Claforan 1 gram q.8. Proventil nebulizer, Serevent, Flonase, theophylline, and Flovent were continued. Patient takes methylcarbinol for degenerative joint disease and this was continued.

On 12/30/99 patient's dyspnea was slightly better but his white count was 15,000. He still had scattered rhonchi.

On 12/31/99 and 1/01/00 patient continued to slowly improve. Had bilateral expiratory and inspiratory wheezes with poor air exchange.

On 12/31/99 Biaxin was added. When the sputum studies were obtained, Pseudomonas was sensitive to Levaquin. The Claforan and Biaxin were discontinued and Levaquin was changed to 500 mg. p.o.

On 1/2/00 patient's dyspnea appeared to be baseline but he still had significant exercise intolerance. Solu-Medrol was discontinued and patient was started on Prednisone 30 mg q. day.

On 1/3/00, day of discharge, patient's dyspnea was back to baseline and he was ambulating. Patient is going to be discharged home on following meds: Levaquin 500 mg q. day for 7 days, Prednisone 20 mg q. day for 3 days, 10 mg. q, day for t=3 days, 5 mg q. day for three days and then stop, Lasix 40 mg q.day, Restoril 15 mg q. h.s. p.r.n. He is advised to resume his home meds.

37. DX 154 History & Physical (Sikder) 12-29-99

Patient with severe advanced coal workers' pneumoconiosis and chronic obstructive pulmonary disease. Patient is currently oxygen dependent. Patient was steroid dependent in the past but was successfully tapered off from the steroids. Currently, he is being maintained on a combination of Flovent MDI, Serevent MDI, and Theophylline and Albuterol nebulizer and good control. The patient was in his usual state of health until 12/29/99 at which time his dyspnea was significantly worse compared to his baseline. At that time, he refused hospitalization. He was treated and given a dose of Solu-Medrol. He states that he was feeling significantly better with Solu-Medrol treatments. Patient was also treated with Rocephin in the office. Since then the patient has been back to the office twice with similar symptoms but refused hospitalization.

On the day of admission, the patient stated that he felt significantly worse with audible wheeze. He was given Zoponex treatment 1.25 mg with persistent wheeze. His saturation was 89% on room air with oxygen supplementation and nebulization treatment. The saturation rate improved to 96%. At this time the patient agrees to hospitalization. He denies any fever, chills, chest pain. He admits to chest tightness. The cough is productive of scanty yellow sputum.

Past Medical History: As per HPI. Flu vaccine in 1997. Pneumovax in 1997.

Family History: Significant for asthma. There is no lung-related illness.

Social History: Smoker of 50 pack years. He quit 13 years ago. He denies ETOH. He is currently a non-smoker.

Occupational History: History of coal mining 15 years, accumulative surface and underground. He retired in 1984. He denies any other significant occupational exposure.

ROS: Denies any nausea, vomiting or diarrhea, or abdominal pain. He admits to pedal edema, orthopnea, and paroxysmal nocturnal dyspnea. He denies any weight gain. He denies any palpitations, dizziness, diaphoresis. Denies any urinary symptoms. Denies any skin rashes. Denies neurological symptoms. All other systems noncontributory.

Physical Examination: Well-developed, elderly white male in mild-to-moderate respiratory distress.

HEENT: Plethoric with rhinophyma. No jugular venous distention. No adenopathy. The patient is using accessory muscles. No thyromegaly. Trachea is midline.
Heart: Regular rate and rhythm. S1 and S2 normal.
Lungs: Poor exchange bilaterally. Rhonchi in both expiratory and inspiration. Prolonged expiratory phase.
Extremities: No cyanosis or clubbing.
Chest: Symmetrical with increased AP diameter. Upper airways is normal.

Impression: (1) Chronic obstructive pulmonary disease exacerbation; (2) Acute bronchitis; (3) Coal workers' pneumoconiosis; (4) Chronic respiratory failure; (5) Cor pulmonale; (6) Carcinoma of the prostate.

The patient will be admitted to the hospital for IV Solu-Medrol and Claforan. He will be continued on aggressive bronchodilator treatment.

38. DX 155 Admitted Central Baptist Hospital (Thompson) 8-28-00

Admitting Diagnosis: COPD, Hematoma, Flank, DIC

39. DX 155 History and Physical Examination (Gerhardstein) 8-28-00

Problem List: (1) Soft tissue bleeding flank, lower abdomen and upper back; (2) Endstage chronic obstructive pulmonary disease (one pack per day for 50 years, quitting in 1985; oxygen dependent for two years; steroid dependent since 11/99); (3) steroid induced myopathy (bedridden since 4/00); (4) history of respiratory failure, pneumonia, and sepsis 4/00; (5) steroid induced diabetes mellitus; (6) prostate cancer diagnosed 6/99; (7) large hiatal hernia with history of reflux; (8) coal workers' pneumoconiosis; (9) Diabetes mellitus; (10) status post right cataract extraction; (11) status post Groshong catheter, 4/00; (12) history of colonoscopy and polypectomy; (13) history of possible atrial arrhythmia on Lanoxin (admission EKG at Highlands with sinus rhythm); (14) elevated alkaline phosphatast (abnormal CT scan of the liver); (15) allergy to Atrovent which causes headache.

Subjective: Chronically ill 76-year-old white male who was hospitalized at Highlands on 8/24/00 with weakness, anorexia, and flank hematoma. Has known severe obstructive lung disease and coal workers' pneumoconiosis and has been on chronic steroids resulting in a myopathy and him being bedridden since 4/00 when he was hospitalized for pneumonia, sepsis, and respiratory failure. Apparently during that admission he was on the ventilator for 72 hours. He was re-hospitalized at the end of 5/00 ad then early 6/00 briefly for an exacerbation. At that time on two liters blood gases revealed pH 7.4, pCO2 46, and pO2 84. He received nebulized bronchodilators, IV steroids ad Claforan with improvement. He states that he has a productive cough of scant sputum that is at his baseline. He wheezes on a daily basis regardless of his steroids. He tends to stay on about 15-20 mg of Prednisone daily. He denies indigestion symptoms since starting Prilosec last winter, 20 mg twice a day. He previously smoked one pack of cigarettes per day for 50 years, quitting in 1985. He retired form the miens in 1984 secondary to shortness of breath. He wants life support and to be full resuscitation at present.

Social History: No alcohol use. Retired miner as noted.
Family History: Mother died at 54 from asthma. One brother died from cancer (a large lesion on left thigh).

Physical Exam:

Vital Signs: Respiratory rate 18. Afebrile.
Heart: Irregular. No S3 gallop.
Lungs: Diffuse mid and end expiratory wheezing. No rales.
Extremities: Trace pedal pulses. No pitting edema. Muscle wasting but no fasciculations.

Lab and X-Ray Data – this is all from Prestonburg:
EKG: Sinus tachycardia. Chest x-ray: No acute infiltrate. Soft tissue swelling in left thorax. Troponin is negative.

Impression: (1) Bleeding and thrombocytopenia, unknown etiology, but requiring extensive transfusion. No preceding trauma and no retroperitoneal bleed by CT scan of the abdomen done in Prestonburg; (2) Endstage emphysema with active bronchospasm but no respiratory distress. Pulse oximetry 98% on two liters; (3) EKG here with atrial fibrillation, rate controlled on Digoxin; (4) History of prostate cancer diagnosed a year ago, untreated; (5) Large hiatal hernia with reflux symptoms controlled by Prilosec; (6) Dizziness rule out CNS Bleed.

40. DX 155 Discharged Central Baptist Hospital (Thompson) 9-5-00

Discharge diagnosis: (1) Metastatic prostate cancer; (2) Subsequent thrombocytopenia; (3) Anemia; (4) Massive hematoma without evidence of retroperitoneal hematoma; (5) Endstage chronic obstructive

pulmonary disease (oxygen dependent for 2 years; steroid dependent since 11/99); (6) Steroid-induced myopathy (bedridden since 4/00); (7) History of reduced respiratory failure/pneumonia/sepsis, 4/00; (8) Steroid-induced diabetes mellitus; (9) History of hiatal hernia with esophageal reflux (on Prilosec); (10) Coal workers' pneumoconiosis; (11) Diabetes mellitus; (12) S/P right cataract extraction; (13) S/P Groshong catheter placement 4/00; (14) History of colonoscopy and polypectomy; (15) History of possible atrial arrythmias on Lanoxin; (16) Elevated alkaline phosphatase level secondary to above; (17) History of allergy to Atrovent which causes headaches; (18) Vertebral fracture.

Patient did not desire extensive resuscitative measure – was considered to be a "limited do not resuscitate" during this hospital stay even though full do not resuscitate status was considered most appropriate, given his metastatic prostate CA and endstage lung disease.

History of Present Illness: Patient has endstage chronic obstructive pulmonary disease. Transferred to Central Baptist from Highlands Regional Medical Ctr. Was admitted to Highlands five days prior to admission after developing left flank hematoma. Hematoma enlarged and he received platelets and blood transfusions. He was treated with inhaled bronchodilators.

Note, he is also being treated for prostate cancer. This was apparently diagnosed by biopsy and he received what was probably Lupron injections.

Hospital Course: Patient was admitted and felt not to have DIC but anemia and thrombocytopenia of other etiology. Subsequently underwent bone marrow biopsy, which confirmed the presence of metastatic prostate cancer. He was treated with Premarin, oral steroids, inhaled bronchodilators, and antibiotics. He received pain control with Oxycodone, Roxanol p.r.n. He subsequently improved and was discharged home in improved and stable condition, although his long-term prognosis is quite poor.

41. DX 155 Hematology/Oncology Consultation (Eldridge) 8-28-00

Focus is on prostate cancer. However, mentions Frasure's complicated lung disease, including COPD and coal workers' pneumoconiosis. Mentions that patient has had respiratory problems in the last couple of years including an episode with respiratory failure requiring mechanical ventilation with steroid myopathy following that. Has also had steroid induced diabetes. Mentions that patient quit smoking 15 years ago and is retired from working in the coal mines. Patient's mother died at age 54 from asthma and pneumonia. Has had some cough with sputum which is occasionally purulent. No pleuritic chest pain or audible wheezing.

42. EX 1 Medical Report (Broudy) 5-12-03
B-reader, Board-certified in internal medicine and pulmonary disease

Opinion: Saw Frasure on two occasions: October 12, 1993 and October 1, 1985. On last occasion, Frasure was 69 and gave a smoking history of about a pack per day for 50 years until he quite about 8 years earlier. Frasure worked 14 ½ years in coal mining but only 1 ½ years underground. The rest was driving a coal truck. Worked steadily until he injured his back. Physical exam revealed evidence of obstructive airways disease with tracheal retraction, hyperresonance to chest percussion, diminished aeration throughout the lungs and marked expiratory delay with wheezing on forced expiration. Spirometry showed evidence of severe obstruction with some restriction of the vital capacity. Blood gases showed mild resting hypoxemia Chest x-rays negative for pneumo. Frasure said he stopped work in 1984. Results of Frasure's 1985 evaluation were similar to the above. Death cert indicates that he died at 77 on October 29, 2001 from respiratory failure due to chronic obstructive pulmonary disease. Had also been previously diagnosed as having widely metastatic prostate cancer with positive bone marrow. Had been hospitalized just 5 days prior to his death because of continued fever, weakness, and increasing confusion.

Based on his review of the entire body of evidence, he did not find that Frasure had pneumoconiosis and it did not cause disability or death. Frasure clearly did not retain the respiratory capacity to perform hard manual labor because of severe chronic obstructive airways disease due to cigarette smoking. He had a long heavy history of smoking. He showed evidence of disability as far back as 1985 or before. There was

no evidence that he had pulmonary disability arising from his occupation as a coal worker. Disagrees with Dr. Sikder's deposition of August 2, 2001. In her deposition, she attributes the impairment to Frasure's exposure to coal dust even though the x-rays were largely read as negative for pneumoconiosis. Certainly, one would not expect to see such severe impairment due to pneumoconiosis unless one had far advanced complicated coal workers' pneumoconiosis, which was clearly not the case. Even the positive interpretations showed only early simple pneumoconiosis. Furthermore, the patient had primarily obstructive impairment which is not the classic type of impairment associated with disabling coal workers' pneumonconiosis.

43. **EX 2** **Medical Report (Dahhan)** **5-12-03**
B-reader, Board-certified in Internal Medicine and Pulmonary Disease Specialist

Opinion: Based on his review of patient's medical records, concluded within a reasonable degree of medical certainty that: (1) Frasure had chronic bronchitis and emphysema; (2) he was disabled prior to his death because of his severe chronic bronchitis and emphysema; (3) he smoked a pack per day for 50 years; (4) he worked in the mining industry for 14 years ending in 1984; (5) there was no radiological data to support a diagnosis of coal workers' pneumoconiosis. The majority of readers found his x-ray to be negative for pneumoconiosis. Some read it as being consistent with category 1 simple coal workers' pneumoconiosis. All noted the presence of emphysema; (6) Dr. Sikder's statement that patient's reduction in FVC indicated a restrictive ventilatory defect is incorrect. When the patient has lost so much lung function due to any cause would result [sic] in a reduction in the FVC and hence, many patients with advanced emphysema such as Mr. Frasure has do [sic] indeed demonstrate a reduction in the FVC in addition to the marked reduction in the FEV1. Overall, the pulmonary function studies performed on this individual are all indicative of a severe obstructive ventilatory defect with no evidence of a restrictive ventilatory defect; (7) Mr. Frasure's severe obstructive airway disease has resulted from chronic bronchitis and emphysema. Simple coal workers' pneumoconiosis does not caused [sic] this type of emphysema; rather as the medical literature indicates, it causes a form of emphysema known has [sic] focal emphysema, which is simply a dilation of the alveolar sacs in the area surrounding the coal macule and is not associated with any destruction of the alveolar structure or loss in the mechanics of the respiratory system as Mr. Frasure's pulmonary function studies demonstrates, indicating that he did not suffer from focal emphysema but rather from cigarette induced centriacinar and panacinar emphysema; (8) Mr. Frasure had a severe obstructive ventilatory impairment that is rarely, if ever, seen secondary to early stages of simple coal workers' pneumoconiosis, which some radiologists reported on his x-ray. He did have a history of 50 pack years of smoking, which is known to cause a severe obstructive pulmonary disease and respiratory impairment; (9) Mr. Frasure's congestive heart failure, steroid induced myopathy and depression are all manifestations of his advanced chronic bronchitis and emphysema that was caused by his lengthy smoking habit and not related to the inhalation of coal dust or coal workers' pneumoconiosis.

44. **EX 3** **Medical Report (Fino)** **5-23-03**
B-reader, Board-certified in Internal Medicine with subspecialty in pulmonary disease

Opinion: Insufficient objective medical evidence to justify diagnosis of coal workers' pneumoconiosis. There was a disabling respiratory impairment present. From a respiratory standpoint, Frasure was disabled from returning to his last coal mining job or a job requiring similar effort. Coal workers' pneumoconiosis played no role in his disability. Even if we were to assume that Frasure had coal workers' pneumoconiosis, it did not contribute to his disability (i.e. would have been disabled had he never set foot in the mines). Death was due to prostate cancer. Coal workers' pneumoconiosis neither caused nor hastened death (i.e. he would have died as and when he did had he never stepped foot in the mines).

45. **EX 4** **Deposition Dahhan** **7-7-03**
B-reader, Board-certified in Internal Medicine and Pulmonary Disease Specialist

Information he reviewed covered period from August 1985 to October 29, 2001. (EX 4: 4). Personally examined Frasure in September 1993. (EX 4: 4). Based upon his review of the information, opined that there was no evidence of coal workers' pneumoconiosis. (EX 4:5).

Had the benefit of numerous spirometric studies, all of which revealed severe obstructive ventilatory defect with various response to bronchodilator therapy. (EX 4:5). This meant that Frasure had an obstructive impairment, which was not completely fixed but rather waxed and waned and produced various responses to the administration of medication. (EX 4:5-6).

Had benefit of arterial blood gas study results. These revealed a significant abnormality. Opined that Frasure had a severe disability, which was a result of a severe obstructive airway disease. Frasure, therefore, would not be able to perform hard manual labor. (EX 4:6).

Recorded Frasure as having been a heavy smoker, averaging 50-pack years. (EX 4:7). Testified that there were sufficient facts in the record (i.e. sufficient occupational and medical history as well as social history, multiple clinical examinations, x-rays, physical examinations, pulmonary function studies, and blood gases) to distinguish between pulmonary disability caused by inhalation of coal versus that caused by cigarette smoke. (EX 4:7). In this case, opined that Frasure's pulmonary disability was due to his 50-pack years of smoking. (EX 4:7).

Reasoning for above opinion: First, Frasure had significant obstructive abnormality that was causing severe disability with very severe reduction in FEV1, which is a parameter of obstructive ventilatory defect. (EX 4:7-8). This type of finding is not seen secondary to inhalation of coal dust. (EX 4:8). Second, Frasure had significant alteration in his blood gas exchange mechanism, indicating that his obstructive defect is severe to alter his to such a major degree. [sic] (EX 4:8). Finally, he had no radiology degree of coal workers' pneumoconiosis of simple or complicated. (EX 4:8). Even if Category 1 simple pneumoconiosis were assumed, it still would not account for the significant severe obstructive defect that's been recorded by all physicians. (EX 4:8).

Also noted that Frasure suffered from chronic bronchitis and emphysema. (EX 4:8). Opined that the cause of these diseases was Frasure's 50-pack years of smoking. (EX 4:8).

Commented on Sikder's deposition: Agrees with Sikder that Frasure had disabling respiratory impairment. (EX 4:9). Agrees that patient was not able to work. (EX 4:9). Disagrees that the patient had a restrictive ventilatory defect because Sikder based that finding on invalid functions. (EX 4:9). Also disagrees that Frasure's pulmonary disability is due to inhalation of coal dust. (EX 4:9).

Testified that Frasure would have had the same type of pulmonary disability even if he never worked in the coal mines. (EX 4:9). Even assuming Frasure suffered from Category 1 pneumconiosis, his opinions would not change because such a radiological diagnosis is not usually associated with such a severe respiratory impairment of the type Frasure had. (EX 4:9).

Admits that some medical literature documents that coal mine dust exposure has a causal effect upon the development of chronic obstructive pulmonary disease. (EX 4:12). Admits that some suggest that an individual might lose some FEV1, which is a parameter of lung function, as a result of exposure to coal dust. (DX 4:12). If accepted, that data indicates that the individual loses about three to five CC's of his FEV1 per year of exposure to coal dust. (EX 4:12). That amount is very minute in an individual's overall respiratory picture. (EX 4:12). Agrees with the literature for the sake of discussion. (EX 4:12). Also testifies that he does not believe that only advanced or complicated pneumoconiosis or progressive massive fibrosis causes disability. (EX 4:12). States that simple coal workers' pneumoconiosis can be the cause of significant lung impairment. (EX 4:13).

Testified that he does not think Category 1/0 pneumoconiosis can cause significant obstructive lung disease such that it would be disabling. (EX 4:16).

46. EX 5 Deposition Fino 7-10-03

B-reader, Board-certified in Internal Medicine with subspecialty in pulmonary disease

Opined that there was no evidence of coal workers' pneumoconiosis. (EX 5:5). Had the benefit of spirometric studies which clearly showed an abnormality – an obstructive defect. (EX 5:5). Opined that the obstruction was due to cigarette smoking. (EX 5:5).

The resting blood gases in the arterial blood gas studies prior to his many hospitalizations (beginning in about 2000) were generally normal. (EX 5:6). There were a couple of very abnormal blood gases prior to 1993, but any resting hypoxemia that he had came back and reversed. (EX 5:6). So blood gases that showed hypoxemia in 1985 no longer showed hypoxemia in 1993. (EX 5:6). This is significant because if one has a coal dust related disease that's causing hypoxia, which can happen, then the hypoxia is present at all times. (EX 5:6). It doesn't come and go. (EX 5:6).

In addition, on two exercise studies in 1993 he did not drop his blood oxygen level with exercise, which again indicates the unlikeliness that coal mine dust contributed to Frasure's respiratory impairment or disability. (EX 5:6). On 12/29/99, Frasure actually maintained a room air arterial blood gas that was normal with a pO2 of 79. (EX 5:6).

Starting on or about 2000, Frasure had numerous problems that caused him to be admitted to the hospital. (EX 5:6). At this time, Frasure's blood gases started to get worse. (EX 5:6). Notes that Frasure was taking a number of medications at this time, all but one of which were being used for obstructive lung disease. (EX 5:7). The obstruction that was being treated was for obstruction due to smoking; it is true that coal mine dust can cause obstruction but that's not treatable. (EX 5:7). Pneumoconiosis is a permanent, irreversible condition and medications are not beneficial in treating coal mine dust related diseases. (DX 5:7).

Opined that Frasure did not retain the respiratory, physiological capacity to perform his past coal mine work. (EX 5:7). Stated that Frasure had a smoking history anywhere from 50 to 100 pack years. (EX 5:8). Testified that there sufficient facts in the record to distinguish that Frasure's pulmonary disability was caused by cigarette smoking rather than coal mine dust. (EX 5:9). These factors include: the pattern of abnormality in the lung function studies and the type of change in the blood gas system. (EX 5:9). Testified that smoking caused Frasure's chronic obstructive pulmonary disease with exacerbation, bronchitis, and cor pulmonale. (EX 5:10).

Disagreed with Sikder's conclusions. (EX 5:10). Disagreed that coal workers' pneumoconiosis was present. (EX 5:11). Stated that smoking and coal dust related lung diseases have similarities but their effects can be separated based on the total amount of coal dust estimated to be inhaled versus the total amount of cigarettes smoked. (EX 5:11). The chest x-ray is helpful as one factor to be considered. (EX 5:11).

Another factor is that toward the end of his life, Frasure began to have elevations in his carbon dioxide level – a condition called hypercarbia. (EX 5:11). One does not expect to see this in pneumoconiosis, unless it was a case of severe scarring and fibrosis as is present in complicated pneumoconiosis. (EX 5;11). Frasure would have had the same type of pulmonary disability had he never been employed in the coal mining industry. (EX 5:11).

In his report stated that Frasure's death was due to prostate cancer. (EX 5:22). Believes that Frasure's breathing impairment did contribute to/hastened his death. (EX 5:23).

47. EX 6 **Deposition Broudy** **6-10-03**
B-reader, Board-certified in internal medicine and pulmonary disease

Examined Frasure on two occasions: October 12, 1993 and October 1, 1985. (EX 6:6). His examinations consisted of pertinent history, physical examination, spirometry, arterial blood gas, and chest x-rays. (EX 6:7).

Opined that Frasure did not have pneumoconiosis nor did it cause disability or death. (EX 6:8). Had the benefit of spirometric studies, which revealed severe chronic obstructive airways disease. (EX 6:9). Arterial blood gas study results revealed mild hypoxemia. Other blood gas studies showed mild or moderate hypoxemia. (EX 6:9). Opined that Frasure did not retain the respiratory capacity to perform hard manual labor. (EX 6:8).

Recorded smoking history of about a pack per day for 50 years. (EX 6:8). Testified that there sufficient facts in the record to distinguish that Frasure's impairment or disability is the result of chronic obstructive pulmonary disease from cigarette smoking rather than coal mine dust. (EX 6:10). First, Frasure had a long significant history of smoking at a rate of a pack per day for 50 years, which is certainly sufficient to cause chronic obstructive airways disease. (EX 6:10). Second, Frasure had the typical impairment associated with smoking: that is chronic airways obstruction as opposed to any type of restrictive defect which one might expect to see with disabling impairment due to pneumoconiosis. (EX 6:10). When causing disabling impairment, pneumconiosis usually is in the complicated form and it causes primarily a restrictive defect. (EX 6:10-11). Neither of those situations were present so that one could reasonably exclude the possibility of coal workers' pneumoconiosis or silicosis causing Frasure's impairment. (EX 6:11).

The x-rays were negative for pneumoconiosis according to his examinations so that one would not expect Frasure to have disabling impairment due to coal workers' pneumoconiosis. EX 6:11).

Noted that the death certificate indicates that Frasure died from respiratory failure due to chronic obstructive airways disease. (EX 6:11). He was also known to have widely metastatic prostate cancer with bony metastatis. (EX 6:12). Based on the fact that Frasure had been hospitalized five days prior to his death, it appears that he died of complications of his prostate cancer and chronic obstructive airways disease. (EX 6:12).

Disagreed with Sikder's conclusions. (EX 6:12). Sikder attributed Frasure's impairment to coal dust exposure even though the x-rays were largely read as negative. (EX 6:12). In addition, one would not expect to see such impairment due to pneumconiosis unless one had far advanced complicatred coal workers' pneumoconiosis. (EX 6:12). Moreover, the primary defect on spirometry was obstructive, which is typical of the COPD from cigarette smoking and not from coal dust. (EX 6:13).

Prior Medical Evidence

With regard to whether there has been a mistake in determination of fact, I must consider all the evidence of record. This evidence is summarized below.

X-Ray Interpretations

Exhibit No.	Date of X-Ray	X-Ray Interpretations and Physician Qualifications	Diagnosis/History Noted Comments
DX 25	8-2-93	Sargent "B/BCR"	No pneumoconiosis
DX 26	8-2-93	Halbert "B/BCR"	No pneumoconiosis
DX 27	8-20-85	Felson "B/BCR"	No pneumoconiosis
DX 28	8-20-85	Wiot "B/BCR"	No pneumoconiosis
DX 29	8-20-85	Spitz "B/BCR"	No pneumoconiosis
DX 30	10-1-85	Quillin "B/BCR"	No pneumoconiosis
DX 31	6-28-93	Lin	p/s/ 2/1
DX 32	6-28-93	Sundaram	p/s 2/1
DX 33	9-25-93	Dahhan "B"	No pneumoconiosis
DX 34	10-12-93	Broudy "B"	No pneumoconiosis
DX 35	10-12-93	Dineen "B, BCR"	No pneumoconiosis

DX 47	9-25-93	Broudy "B"	No pneumoconiosis
DX 48	9-25-93	Jarboe "B/BCR"	No pneumoconiosis
DX 49	9-25-93	Dineen "B/BCR"	No pneumoconiosis
DX 50	3-11-85	Marshall "B/BCR"	p/p 1/0 with associated emphysema
DX 50	6-20-85	Ameji	2/1 p/q with multiple s type of opacities in both lungs
DX 50	8-1-85	Lagada	1/1 p/q
DX 50	6-19-95	Myer "B"	1/0 p/p, all zones both lungs
DX 50	10/15/85	Bangudi	1/1 s
DX 50	---	Wright	1/0 q/p
DX 50	6-19-85	deGuzman	Noted bilateral pleural thickening and scattered fibrodoular densities in both lung fields, suggestive of 1/2 p/q
DX 50	10-21-85	El-Amin	1/1 p/q
DX 50	3-11-85	Brandon "B/BCR"	1/0 p/p
DX 51	10-1-85	Broudy "B"	No pneumoconiosis
DX 52	9-25-93	Dahhan "B"	Chest x-ray showed changes consistent with emphysema with bilateral bullae formation. Otherwise lung fields are clear with no pleural or parenchymal abnormalities consistent with pneumo being present. Classified as 0/0.
DX 57	6-28-94	Sundaram	s/s 1/1
DX 58	6-28-94	Bassali "B/BCR"	q/t 1/1
DX 58	6-28-93	Bassali "B/BCR"	p/s 1/1
DX 61	6-28-93	Broudy "B"	No pneumoconiosis
DX 63	6-28-93	Jarboe "B/BCR"	No pneumoconiosis
DX 64	6-28-93	Sargent "B/BCR"	No pneumoconiosis
DX 64	6-28-93	Barrett "B/BCR"	No pneumoconiosis
DX 64	9-25-93	Sargent "B/BCR"	No pneumoconiosis
DX 64	9-25-93	Barrett "B/BCR"	No pneumoconiosis
DX 65	6-28-93	Dineen "B/BCR"	No pneumoconiosis
DX 66	1-6-95	Myer	Silicosis category 1/0 p/p. both mid and upper lung zones
DX 66	6-28-94	Marshall "B/BCR"	q/p 1/1
DX 67	1-6-95	Marshall "B/BCR"	q/p 1/1
DX 68	9-25-93	Marshall "B/BCR"	q/p 1/1
DX 69	10-12-93	Marshall "B/BCR"	q/p 1/1
DX 70	1-7-95	Wright	q/p 1/0
DX 70	6-28-94	Brandon "B/BCR"	s/s 1/1
DX 71	10-12-93	Brandon "B/BCR"	q/p 1/1
DX 72	9-25-93	Brandon "B/BCR"	s/p 1/1
DX 73	8-2-93	Marshall "B/BCR"	p/q 2/1
DX 78	10-12-93	Sargent "B/BCR"	No pneumoconiosis
DX 78	6-28-94	Sargent "B/BCR"	No pneumoconiosis
DX 78	1-6-95	Sargent "B/BCR"	No pneumoconiosis
DX 79	2-3-95	Broudy "B"	No pneumoconiosis

DX 80	10-12-93	Binns "B/BCR"	No pneumoconiosis
DX 80	10-12-93	Abramowitz "B/BCR"	No pneumoconiosis
DX 80	10-12-93	Gogineni "B/BCR"	No pneumoconiosis
DX 81	1-6-95	Binns "B/BCR"	No pneumoconiosis
DX 81	1-6-95	Abramowitz "B/BCR"	No pneumoconiosis
DX 81	1-6-95	Gogineni "B/BCR"	No pneumoconiosis
DX 82	2-3-95	Jarboe "B/BCR"	No pneumoconiosis
DX 83	2-3-95	Binns "B/BCR"	No pneumoconiosis
DX 83	2-3-95	Wershba "B/BCR"	No pneumoconiosis
DX 84	6-28-94	Abramowitz "B/BCR"	No pneumoconiosis
DX 84	6-28-94	Wershba "B/BCR"	No pneumoconiosis
DX 84	6-28-94	Gogineni "B/BCR"	No pneumoconiosis
DX 85	2-3-95 (Re-read)	Sargent "B/BCR"	No pneumoconiosis
DX 86	6-28-93	Marshall "B/BCR"	q/p 2/1
DX 87	2-3-95	Brandon "B/BCR"	p/s 1/1
DX 88	6-28-94	Baker "B"	p/q 1/0
DX 89	2-3-95	Marhsall "B/BCR"	q/p 2/1
DX 90 DX 97	6-28-94	Wright	Non-specific interstitial opacities consistent with category 1/1 q/q pneumoconiosis, simple
DX 91	6-28-93	Baker "B"	p/p 1/0
DX 91	9-25-93	Baker "B"	p/p 1/0
DX 91	10-12-93	Baker "B"	p/q 1/0
DX 95	1-7-95	Wright	1/0 q/p
DX 97	6-28-94	Wright	q/q 1/1

Pulmonary Function Studies

Ex. No.	Test Date	Physician	FEV_1	FVC	MVV	TR	Age/Height	Coop/Comp
DX 8	8-20-85	O'Neill	1.10	2.38	30		61/70 ″	Good coop/comp; fairly good effort
DX 9	6-28-93	Sundaram	Pre-B: 0.85 Post-B: 1.08	Pre-B: 1.59 Post-B: 1.79	Pre-B: -- Post-B: 32.6		69/71″	-----
DX 10	8-2-93	Mettu	0.76	2.04	24		69/69″	Good coop/comp
DX 11	9-25-93	Dahhan	Pre-B: 0.93 Post-B: 1.12	Pre-B: 2.36 Post-B: 2.76	Pre-B: 24 Post-B: 33		69/69½″	Good coop/comp
DX 12	10-12-93	Broudy	1.14	2.99	33		69/70″	Effort variable; didn't really give it all; said best he could do
DX 13	10-21-83	Anderson	1.0	31%	28%		59	
DX 66	1-6-95	Myer	Pre-B:	Pre-B:	Pre-B:		71	

			0.94 Post-B: 1.14	2.30 Post-B: 2.54	Post-B:			
DX 70	1-7-95	Wright	0.83	2.02			70/69″	
DX 77	2-3-95	Vuskovich	Pre-B: 1.01 Post-B: 1.32	Pre-B: 2.35 Post-B: 3.17				
DX 134	11-4-98	Sikder	0.75	1.70			74/71″	

Arterial Blood Gas Studies

Exhibit No.	Date	Physician	PCO_2	PO_2	Comments
DX 12					
DX 13	10-21-83	Anderson	41	66	
DX 14 DX 22	8-20-85	O'Neill	37.1	56.9	Moderately severe hypoxemia
DX 19	10-12-93	Broudy			Mild resting arterial hypoxemia
DX 20 DX 23	8-2-93	Mettu	42.4 (at rest) 41.4 (exercise)	71.4 (at rest) 81.4 (exercise)	
DX 24	9-25-93	Dahhan	41.5 (at rest) 42.4 (exercise)	70.7 (at rest) 77.7 (exercise)	
DX 70	1-7-95	Wright	45	72	This represents mild resting arterial hypoxemia

Relevant Examination, Medical Reports and Depositions

	Exhibit No.	***Physician and Qualifications***	***Exam/Report Date***
1.	DX 13	Anderson Board-certified internal medicine and pulmonary disease	12-14-83

Comments: Dr. Anderson submitted a medical report based on a physical examination, which included a chest x-ray, blood gas testing, and pulmonary function studies. The examination was performed on 10-21-83. At the time of the exam, Frasure had still been working as a miner, however, he had been short of breath for about two years. He could walk more than ½ mile on level and climb 2 flights of stairs. Over the last two years, he had been awakened an average of 2-3 times per week at night by shortness of breath. He had a cough for the last 3-4 years that may be productive of more than two tablespoons of sputum. He had some chest pain described as soreness, made worse with exertion or when he had to breathe dust. The pain was relieved by resting or not breathing dust. Recorded cigarette smoking history of ¾ of a pack per day since miner was a child. He had lost over 25 lbs. over the prior year. He did not take any medications, nor did he have any operations. Chest x-ray revealed bilateral emphysema but no evidence of pneumoconiosis.

Conclusions: Found miner to be suffering from pulmonary emphysema with severe obstructive ventilatory insufficiency. Found no evidence of pneumoconiosis. Noted that this represents significant decrease in pulmonary function over last three years and miner is now at a level where he would be eligible for social security disability benefits.

2. DX 14 O'Neill 8-27-85

Comments: Dr. O'Neill submitted a medical report based on a physical examination, which included a chest x-ray, blood gas testing, and pulmonary function studies. The examination was performed on 8-20-85. Miner stated that for the past two or more years he has been troubled with progressive exertional wheezing dyspnea. At that time, he stated that he was unable to walk more than one block on the level or climb up more than five to six steps without getting unduly short of breath. He denied orthopnea. For the past six months, he had experienced occasional episodes of paroxysmal nocturnal dyspnea which was relieved by getting up and expectorating. For the pat two or more years, he had a chronic productive cough. He denied hemoptysis. For the past six to eight months, he had experienced left upper anterior chest pain which was intermittent, sharp, and which had no radiation or relationship. He denied a previous history of pneumonia, pleurisy, asthma or tuberculosis. He denied a previous history of cardiac disease or hypertension. In terms of his respiratory system, there was no peripheral cyanosis and no digital clubbing. Flow velocity was markedly decreased. Percussion note hyperresonant. Aeration was significantly diminished. The breath sounds were coarsely bronchovesicular in quality and an early phase expiratory wheeze was elicited. Recorded a smoking history of 2/3 of a pack daily for most of miner's adult life. Based upon his exam, he concluded miner was suffering from severe obstructive airway disease with restrictive component, chronic bronchitis, probably emphysema, and a history of low back injury. No digital clubbing. Chest x-ray: PA and lateral, 8-20-85. The x-ray was of good diagnostic quality. Bony thorax showed wedging of D11. Outline of heart and great vessels was within normal limits. Lungs were hyperinflated. Calcified granuloma was seen in left and mid lung zone. Lung fields were free of infiltrative lesions. Impression: Hyperinflation consistent with obstructive airways disease. UICC 0/0. Pulmonary Function Studies: Spirometry showed severe obstructive airway disease with a restrictive component. Arterial blood gas study showed moderately severe hypoxemia with a p02 of 56.9 mmHg and a pCO2 of 37.1 mmHg.

Conclusions: Severe obstructive airway disease with restrictive component. Chronic bronchitis. Probable emphysema. No evidence of pneumoconiosis. History of low back injury.

3. DX 15 Broudy 10-3-85
B-reader, Board-certified in internal medicine and pulmonary disease

Comments: Dr. Broudy submitted a medical report based on a physical examination, which included a chest x-ray, blood gas testing, and pulmonary function studies. The examination was performed on 10-1-85. Recorded smoking history of ¾ of a pack per day for about 50 years. Had breathing trouble for a couple of years. Complained of constant rattling in chest and chronic congestion which would not clear. He had trouble climbing up steps to his house or getting into his truck. He coughed a lot and raised clear or gray-colored phlegm. There was no hemoptysis. He had occasional pain in upper chest which last only a few seconds. There was almost constant wheezing. He had dyspnea on exertion walking short distances and had to go slowly up stairs. No history of edema. No history of tuberculosis, carcinoma, asthma, pneumonia, stroke, peptic ulcer disease, heart attack, kidney stones, diabetes, or hypertension. Medications included a breathing medication and pain pills. Chest is hyperresonant to percussion. There is decreased chest expansion. There was very poor aeration of the lungs. There was severe expiratory delay with diffuse expiratory wheezing. There was no cyanosis, clubbing, or edema of the extremities. Arterial bllod gas study was virtually normal. Spirometry showed evidence of severe obstructive airways disease with marked restriction of the vital capacity. Good effort made by patient on spirometry. Chest x-rays were of good diagnostic quality. Lungs zones were clear except for a calcified granuloma in the lingual. No evidence of pneumoconiosis – categorized film as Category 0.

Conclusions: He diagnosed chronic bronchitis, and pulmonary emphysema with very severe chronic obstructive airways obstruction. Quote: "I do not believe Mr. Frasure has coal workers' pneumoconiosis. Because of his severe respiratory impairment due to chronic bronchitis and emphysema, I do not believe he has the respiratory functional capacity to perform the work of an underground coal miner. I believe the chronic bronchitis and emphysema us a result of cigarette smoking. I do not believe there has been any significant pulmonary disease or respiratory impairment which has arisen from this man's occupation as a coal miner."

4. DX 16 Bryson 11-4-85

Comments: Documented smoking history of ¾ of a pack per day for 50 years. Noted cough, shortness of breath. Distance producing dyspnea was 1 block.

Conclusions: Pneumoconiosis stage zero. Noted that patient may return to underground coal mining.

5. DX 17 Sundaram 6-28-93

Comments: Performed physical examination on 6-28-93 that included chest x-ray and pulmonary function testing. Noted that patient experienced shortness of breath on walking a distance of ½ block. Quit smoking 8 years ago. No edema nor clubbing. Chest x-ray: 2/1. Vent Study: (see chart above – DX 9).

Conclusions: Found the chest x-ray to be positive for pneumoconiosis. Based upon his exam, diagnosed coal workers' pneumoconiosis due to prolonged exposure to coal dust. Opined that miner was totally disabled.

6. DX 18 Dahhan 9-27-93
B-reader, Board certified in internal medicine and in pulmonary disease.

Comments: Performed physical examination on 9-25-93 that included chest x-ray, spirometry, and arterial blood gas studies. Recorded a smoking history of one pack per day from age of 10, the miner having quit in 1985. Had a history of daily cough with the production of clear sputum with no hemoptysis. Had frequent wheezing. On an inhaler as needed and Theophylline Liquid. Has dyspnea on exertion, such as a flight of stairs. Has no history of orthopnea, paroxysmal, dyspnea, edema, hypertension, or chest pain. Examination of chest showed increased AP diameter with hyper resonancy to percussion. In auscultation reduced air entry to both lungs were noted with prolongation of the expiratory phase and scattered bilateral expiratory wheeze. No clubbing or edema. Arterial blood gases at rest shoed minimum hypoxia with adequate ventilation with a p02 of 70.7 and pC02 of 41.5. End of exercise, blood gases showed normal oxygen with adequate ventilation with a p02 of 77.7 and pC02 of 42.4. Spirometry showed severe degree of airway obstruction with partial reversibility after the administration of bronchodilators. FVC of 2.36 liter or 52% of predicted. FEV1 of 0.93 liter or 29% of predicted. After bronchodilators: FVC of 2.76 liter or 61% of predicted. FEV1 of 1.12 liter or 33% of predicted. Chest x-ray showed changes consistent with emphysema with bilateral bullae formation. Otherwise, the lung fields are clear with no pleural or parenchymal abnormalities consistent with pneumoconiosis being present. ILO classification is 0/0.

Conclusions: Found insufficient objective evidence for diagnosis of occupational pneumoconiosis based on obstructive abnormality on clinical exam of chest, negative x-ray readings, and obstructive abnormality on pulmonary function studies, and alteration of the blood gas exchange mechanism at rest that correct after exercise. Diagnosed chronic obstructive lung disease of the variety of chronic bronchitis and emphysema as demonstrated by the clinical, radiological and physiological data. Opined that the cause of miner's lung disease was his 40 pack years of smoking. Found the miner to be disabled from a respiratory standpoint,

however, miner did not have evidence of pulmonary impairment and/or disability caused by or contributed to by coal dust exposure or occupational pneumoconiosis.

7. DX 19 Broudy 10-12-93
B-reader, Board-certified in internal medicine and pulmonary disease

Comments: Performed physical examination on 10-12-93 that included chest x-ray, spirometry, and arterial blood gas studies. Recorded smoking history of 50 years, consuming about a pack per day until 8 years ago. Breathing bothered by dust on the job. Miner says that he cannot climb hills and cannot do much walking or anything strenuous because of his breathing trouble. He is on liquid Theophylline preparation and rarely uses a metered dose inhaler. He was once told by Dr. Adams, his regular physician, that he had dust on his lungs. He has had no trouble sleeping or chest pain. He does have frequent wheezing. He has had daily cough and dark foamy phlegm for 10-15 years. There is no history of hemoptysis, weight loss, fever or edema. Past medical history reveals that he was hospitalized for his lungs in 1985 or 1986. Chest is hyperresonant to percussion. Lungs have decreased aeration throughout. No cyanosis, clubbing, or edema of extremities. There is marked expiratory delay with wheezing on forced expiration throughout. Spirometry shows evidence of severe obstructive airways disease with some restriction of the vital capacity. Results are slightly better than what was obtained on 10-1-85. Patient made a good effort. Arterial blood gas study shows mild resting arterial hypoxemia. Total hemoglobin is 15.5 grams. Chest x-rays are of good diagnostic quality. Lung zones are clear except for scattered calcified granulomas. Saw no evidence of coal workers' pneumoconiosis and would categorize the films as Category 0.

Conclusions: Diagnosed chronic bronchitis and pulmonary emphysema with severe chronic obstructive airways disease. Concluded that miner did not have coal workers' pneumoconiosis. Opined that miner was suffering from chronic bronchitis and pulmonary emphysema with severe chronic obstructive airways disease as a result of cigarette smoking. Found miner to be disabled as a result of condition. Did not believe there was any significant pulmonary disease or respiratory impairment which had arisen from coal mine dust exposure.

8. DX 20 Mettu 6-28-93

Comments: Performed physical examination on 8-2-93. Noted a history of cough with mucoid expectoration almost every single day, mostly in the morning for the past 15 years. Gave a history of wheezing since 1986, mostly in humid weather and also at night. Has exertional shortness of breath and cannot climb even 1 flight of stairs. Had pneumonia in 1985. Recorded a smoking history of one pack per day fro 1934 until 1985. Pulmonary function studies: FVC 2.04 liters; 47% of predicted. FEV1 0.76 cc; 22% of the predicted value. FEV1% 37, MVV 24 liters; 19% of predicted value. Severe obstructive airway disease with decreased MVV. Understanding and cooperation were good. Arterial blood gases at rest on room air PH 7.38, PC02 42.4, P02 71.4, % Saturation 93.8. Arterial blood gases after exercise PH 7.40, PC02 41.4, P02 81.4, % Saturation 95.8.

Conclusions: Diagnosed chronic bronchitis. Found miner's impairment to be severe, as evidenced by pulmonary function testing.

9. DX 50 Ameji 6-20-85

Comments: Performed physical examination on 6-20-85. Miner's chief complaint at that time was shortness of breath and difficulty in breathing on mild exertion. Pulmonary Function Studies: FVC predicted was 4.30 liters; measured 1.78 liters. That brings a percentage of 44%. The FEV predicted was 2.82 liters; measured was 0.98. That brings a percentage of 35%. FEV3 was 66% total. The MVV predicted was 127; measured, 17. That brings a percentage of 17%. Arterial Blood Gas Studies Done on 5/1/85: pH was 7.431. The PCO was 38. The PO2 was 67.1, which shows Frasure had marked hypoxemia. That means the oxygen in the blood is greatly reduced. Chest x-ray of good quality showed multiple small

micronodular opacities in both lungs consistent with 2/1 and p/q with multiple s type of opacities in both lungs. Recorded a smoking history of about a pack per day for 50 yrs. Also examined miner regarding back pain, submitting a report dated July 10, 1985 and hospital records from March 29 to April 8, 1985.

Conclusions: Found there to be coal workers' pneumoconiosis. In his opinion, miner was unable to work as a result of his back and lungs and coal mine dust exposure.

10. DX 50 deGuzman 6-26-85

Comments: Performed physical examination on 6-26-85. Miner complained of coughing, productive, and shortness of breath. Reviewed x-ray of good quality that was done in June of 1985. X-ray showed emphysematous-type chest. It had bilateral pleural thickening, and also scattered fibronodular densities in both lung fields, suggestive of pneumoconiosis stage1/2, p/q (simple pneumoconiosis). Noticed clubbing of the fingers on both hands. This denotes decreased oxygenation of blood moving to the lungs.

Conclusions: Pneumoconiosis stage1/2, p/q (simple pneumoconiosis). While admits that other disease entities can produce nodulation upon x-ray that is similar to pneumoconiosis, the nodulation on this x-ray is typical of coal workers' pneumoconiosis.

11. DX 50 Lagada 8-7-85 and 8-14-85

Comments: Performed physical examinations on 8-7-85 and 8-14-85. The miner's complaints were shortness of breath and pain in the low back. Miner had difficulty walking 10 feet. He had paradoxical abdominal reaction to breathing. He had decreased breath sounds with bronchial quality and diminished expansion of the chest wall. Stated that miner's pulmonary studies were indicative of a very severe obstructive and restrictive disease. The results were as follows: FVC: 1.78; predicted, 4.03 and that represents 44%; the FEV1: 0.98; the predicted 2.82 and that is equivalent to 35%; and the MVV: 21, the predicted 127, and that is equal to 17%. Reported that miner had small airway disease. Evaluated chest x-ray of good quality dated 8/1/85 and read it as 1/1 and p/q (early pneumoconiosis). Received a smoking history that Frasure smoked one pack a day – does not know duration.

Conclusions: Diagnosed chronic obstructive pulmonary disease and pneumoconiosis; low back syndrome; peripheral vascular disease. Recommended that patient not go back to the coal mines and/or involve in physical activity requiring lifting of more than five pounds. Stated that miner would be disabled from his lungs alone, even if he did not suffer from back problems. In his opinion, miner was totally disabled as a result of lungs and back. Lagada could not pick out any one certain part of the miner's employment that caused his pneumoconiosis – stated that it all contributed.

12. DX 50 El-Amin 10-21-85 and 10-24-85

Comments: Examined the miner on 10-21-85 and 10-24-85. His chief complaint was cough, sputum production, shortness of breath, and dyspnea on exertion. He was taking bronchodilator Theophyllin for his breathing. Miner had dry rales at the lung bases and decreased chest expansion, which are abnormal findings. Dry rales of the lung bases tends to show interstitial lung disease or alveoli destruction and decreased chest expansion usually seen in people who are up in age. Interpreted chest film of good quality as stage 1/1 p/q. Pulmonary Function Studies: Grossly abnormal in all three perimeters. His MVV was 17% predicted which measures small airway disease. So he has a small degree of airway disease. His FVC measures restrictive lung disease, which is also markedly reduced at 44. His FEV1 was also markedly abnormal at 35%. This would be compatible with his clinical findings of deceased chest expansion and the rales at the lung bases. Blood gases: Also abnormal. His PO2 was markedly diminished at 67. PCO2 was 38.3. All the other perimeters of the arterial blood gas was essentially negative. This shows hypoxemia and is consistent with his complaint of shortness of breath. Recorded smoking history of ¾ of a

pack per day; did not know duration. Testified that there was no way to apportion the pulmonary disability caused by smoking and that caused by pneumoconiosis.

Conclusions: Believes his shortness of breath is a result of his pneumoconiosis (early stage). Miner's entire employment history contributed to his pneumoconiosis – could not pick out one particular part of his employment as the cause.

13.	DX 50	Bangudi A-reader	10-21-85

Comments: Examined patient on 10-21-85. Miner's chief complaint was exertional shortness of breath and productive cough. Miner stated that this started about two years ago and had been getting worse. Miner would wake up in the middle of the night due to smothering. He had chest pain relieved with rest, unrelated to exercise. Denied any history of myocardial infarction, congestive heart failure, hypertension, rheumatic fever, asthma, chronic bronchitis, or tuberculosis. Had no history of cancer. Miner was taking "breathing pill" for six months or longer and Darvocet. Recorded smoking history of about ¾ of a pack per day for about 50 yrs. With regard to chest and lungs, miner had bilateral wheezes and rhonchi at the bases. With regard to extremities, miner had no clubbing, cyanosis, or edema. Had a chest x-ray to review but did not have the benefit of pulmonary function or blood gas studies. Read chest x-ray dated 10/15/85. Noted small irregular opacities in all zones. These changes are compatible with coal workers' pneumoconiosis, ILO Classification 1/1 s.

Conclusions: Opined that miner suffered from pneumoconiosis. Opined that entire exposure history contributed to pneumoconiosis.

14.	DX 50	Wright	No Date Given

Comments: Took x-rays of excellent quality and assigned them a classification of 1/0 q and p. Pulmonary function results: 29% of predicted on the FEV; 50% predicted on the FVC; and 21% predicted on the MVV. Blood gas study results: PO2 at 59.3 and PCO2 at 38.4 and the PH at 7.37. These are abnormal. Opined that entire exposure history contributed to pneumoconiosis.

Conclusions: Diagnosed Category 1 pneumoconiosis. In addition, diagnosed chronic bronchitis, which could also be attributable to many years of exposure to coal dust).

15.	DX 50	Marshall Board-certified in radiology; B-reader	3-11-85

Comments: Interpreted X-Ray dated 3/11/1985 on 4/30/1985. Film was of quality "two" meaning that it was slightly overexposed, but of good quality for the purpose that it was being used. Showed pneumoconiosis p/p , five zones of the lung and a profusion of 1/0, with associated emphysema.

Conclusions: Diagnosed pneumoconiosis. Opined that entire exposure history contributed to pneumoconiosis.

16.	DX 50	Myer Board-certified in internal medicine; B-reader	8-16-85

Comments: Never saw miner personally. Examined x-ray of good quality dated 6/19/85 on 8/16/85. Testified that x-ray shows a PA and lateral chest which reveal a normal bony thorax. Heart size is normal. The costophrenic angles are sharp. The hilar regions are rather prominent. There is a calcified nodule in the left, mid-lower lung zone about six millimeters in diameter. The lung fields, otherwise, show early micronodular opacities compatible with silicosis Category 1/0-p/p, all zones both lungs. Did not perform physical exam or take history from Frasure. Also, did not perform any pulmonary studies.

Conclusions: Blames silicosis on entire exposure history. With history as given, thinks that silicosis in this case is most likely coal workers' type pneumoconiosis, though cannot distinguish silicosis and coal workers' pneumoconiosis radiographically.

17. DX 50 Brandon 10-30-85
Board-certified in radiology;
B-reader

Comments: Examined x-ray of film quality "two" (good quality) dated 3/11/85 on 10/30/85. Read it as 1/0, p/p profusion (one of the earliest stages of pneumoconiosis). Opined that entire exposure history contributed to pneumoconiosis.

Conclusions: Diagnosed pneumoconiosis (early stage). Testified that he was not aware of any study indicating that experts are more apt to disagree regarding the interpretation of x-ray film when earlier stages of pneumoconiosis are at issue.

18. DX 66 Myer 1-6-95

Comments: Miner stated that he had trouble breathing for the past twelve years, in fact, stopped working because of his breathing. He is dyspneic [sic] on walking a half block or climbing a flight of stairs. He coughs day and night with sputa production and wheezes at times. He sleeps on only one pillow and denies chest pain, syncope or edema. He uses Theophyllin for his lungs. He smoked a pack of cigarettes a day for forty-five years, but quit smoking ten years ago. Past illnesses reveal no major operations, injuries, or illnesses. He denies heart trouble, high blood pressure, pneumonia, etc. His only hospitalizations have been for breathing problems.

Miner's chief complaint is his lungs. Patient is a rather asthenic white male who appears in no acute distress, but is obviously short of breath and wheezes audibly. He coughs frequently. Chest expansion is ¾ of an inch at the xiphoid process, being somewhat limited. Distant wheezes are heard throughout with significant impairment if air exchange obvious despite recent use of bronchodilators. No clubbing, cyanosis, nor edema.

Chest x-ray of quality "one" dated 1-6-95 read as silicosis, category 1/0 p/p, both mid and upper lung zones. Vent study results: FVC 2.30 L, 2.14 L; FEV 1 0.94 L, 0.98 L before bronchodilators. FVC 2.45 L, 2.38 L; FEV 1 1.14 L, 1.04 L after bronchodilators. These were done before and after bronchodilators because of significant wheezing and a very low %FEV1. IMP: Severe obstructive defect in ventilation, possible associated moderately severe restrictive defect in ventilation, without significant improvement with use of bronchodilators. Class IV under AMA Guidelines. These would meet the criteria for disability under Federal Black Lung Regulation 718, Appendix .

Electrocardiogram: Slight ST segment straightening in leads II, III, and AVF. Small Q in AVL. Cannot exclude changes of early ischemia. Borderline electrocardiogram.

Conclusions: Silicosis, category 1/0 p/p, both mid and upper lung zones. Chronic obstructive pulmonary disease. This man's respiratory function impairment and x-ray changes are most likely related not only to his coal dust exposure, but to his exposure to dust in his work with the highway department.

19. DX 70 Wright 1-7-95

Comments: Examined miner on 1-7-95. Miner's chief complaint was shortness of breath. He has had these symptoms for the last 10 to 12 years. He also complains of a productive cough, bringing up one cup of brown phlegm each day. He denies hemoptysis. He claims wheezing which is frequent, occurring both at rest as well as with exertion, sometimes relieved by medication. He presently takes a bronchodilator (Theophylline). He smoked one pack of cigarettes daily for 40 years, but says he stopped ten years ago and has not smoked since. He denies chest pain. No history of heart disease, hypertension, diabetes, tuberculosis or other major medical illnesses. He was hospitalized in 1984 for shortness of breath. No major surgeries.

Examination confined to the chest. Ausculation of the lung fields reveals bilateral expiratory wheezing; breath sounds were distant. There was an increased diameter of the chest. No finger clubbing, cyanosis or ankle edema. Interpreted x-ray dated 1-7-95. There are PA and lateral views of the chest of excellent quality. There is interstitial nodulation seen in all lung zones. Classified as 1/0 q/p – em. The lung fields are hyperinflated and there are scattered granulomatous. This profusion rating may be underread because of patient's emphysema.

Vent Study: Best FVC 2.02, predicted 46%, second best is 1.89%. Best FEV1 0.83, % predicted 25%, second best 0.73%. FEV1/FVC ratio is 41%. These findings indicate a severe obstructive impairment as well as a restrictive disease. This is an abnormal study (i.e. the patient could have a high reading of pneumoconiosis than interpreted indicating marked pulmonary impairment).

Arterial Blood Gas: PO2 72, PCO2 45, pH 7.36. This represents mild resting arterial hypoxemia.

Conclusions: Non-specific interstitial opacities consistent with category 1/0 pneumoconiosis, simple. No acute abnormalities seen. Chronic obstructive pulmonary disease with mixed bronchitic and emphysematous types; severe. Old healed granulomatous disease. Etiology of pulmonary impairment is mixed related to both dust inhalation and smoking. Although the patient appears to have simple pneumoconiosis at an early stage on the x-ray his pulmonary function studies and physical findings would indicate a much more severe impairment. The patient cannot perform the ordinary work of a coal miner. He is disabled for all but sedentary activities.

20. DX 77 Vuskovich 2-3-95 (exam)
2-13-95 (report)

Comments: Examined patient on 2-3-95. Patient's chief complaint was exertional dyspnea that started in 1983. Patient stated that it is not getting worse. It is not seasonal but is characterized by exacerbations and remissions. Other complaints consist of a wheeze with exertion and with recumbency. He denies cough, chest pain, pedal edema, orthopnea, paroxysmal nocturnal dyspnea and hemoptysis. He denies a history of asthma, emphysema, chronic bronchitis, tuberculosis, pleurisy, pneumonia, heart trouble of any kind and allergic rhinitis. Started smoking as a child – up to four packs per day – and quit in 1985. There is a strong family history of asthma; there is no family history of emphysema, coronary heart disease, or diabetes.

Audible wheeze. No cough, labored breathing, hoarseness, cyanosis, nor pallor. With regard to chest, the breath sounds were distant and there was wheeze throughout both lung fields. There was poor lateral excursion of the lower ribs. With regard to extremities, there was no clubbing, pedal edema, nor limp.

Pulmonary Function Studies: He made a good effort to generate valid studies. Pre-bronchodilator studies, FVC 2.35 L, 58% of predicted; FEV1 1.01 L, 32% of predicted. Ratio is 43%. Post-bronchodilator pulmonary function studies, FVC 3.17 L, 79% of predicted; FEV1 1.32 L, 42% of predicted. Ratio is 42%. Interpretation: Pre-bronchodilator studies revealed a severe obstructive impairment. Post-bronchodilator revealed moderate obstructive impairment. Significant improvement with bronchodilator therapy.

12-lead electrocardiogram – low voltage in limb leads. This finding is consistent with emphysema.

Took EPA and lateral chest x-ray of quality "one" dated 2-3-95. Examination of lung fields show evidence of bullae and emphysema as well as hyperinflation. Impression: Chronic obstructive emphysema.

Conclusions: Diagnosis: (1) Chronic obstructive emphysema; (2) Severe obstructive impairment secondary to chronic obstructive emphysema. I would apportion 100% of his impairment to chronic obstructive emphysema secondary to cigarette abuse. He does not have an occupational pulmonary disease. It is obvious he has not reached maximum medical improvement. With proper therapy he would have improvement in his pulmonary function.

Valid pulmonary function studies revealed a severe obstructive impairment secondary to emphysema. The impairment is in no way related to his occupation in the coal industry. From a physical standpoint, he would be unable to return to his regular coal mining job or work requiring similar physical effort.

21. DX 21 Mettu 11-12-93

Opinion: Stated that miner had symptoms of chronic bronchitis. Pulmonary function studies revealed FVC 47% of the predicted value. FEV1 22% of the predicted value. Arterial blood bases PO2 71.4, with exercise PO2 81.4. Miner was permanently and totally disabled due to respiratory impairment. Etiology factors include cigarette smoking and "also he did work in the coal mines." Also stated: "There could be a complaint of coal dust exposure for his chronic bronchitis and obstructive airway disease." Also stated that the miner had sufficient time to contact coal workers' pneumoconiosis, however, x-ray evidence was negative, which was read by B-readers. Opined that it is very difficult to differentiate between pulmonary impairment caused by pneumoconiosis and that caused by smoking. Miner had cough with mucoid expectoration of chronic bronchitic type and had abnormal pulmonary function studies and arterial blood gases. Unable to differentiate definitively between smoking and coal dust but found it possible that coal dust exposure was partially responsible for the miner's pulmonary impairment. If he did have pneumoconiosis, found that it would have contributed to his respiratory impairment.

22. DX 59 Branscomb 9-7-94
B-reader, Board-certified in Internal Medicine

Opinion: In the case of Mr. Frasure, there is ample demonstration that he does not have restrictive physiology but only obstructive: the x-rays showed hyperinflation according to many readers. The physical signs of obstruction were commonly found, namely wheezing.

With a high level of medical certainty, found that miner did not have coal workers' pneumoconiosis or other occupational pulmonary disease and no pulmonary impairment secondary to inhalation of dust in or around coal mines. The records establish that from a pulmonary point of view, he is not capable of continuing his previous work in coal mining. All of his impairments are those of the general population. None are caused or influenced by coal mine dust exposure. His impairments are the result of severe chronic obstructive pulmonary disease and a severe back injury. The COPD was caused by tobacco abuse and neither caused nor aggravated by coal dust exposure.

23. DX 60 Fino 9-15-94
B-reader, Board-certified in Internal Medicine with subspecialty in pulmonary disease

Opinion: Opined that Frasure did not suffer from an occupationally acquired pulmonary condition as a result of coal mine dust exposure. Believed this to be the case because: (1) the majority of chest x-ray readings were negative for pneumoconiosis; (2) the spirometric evaluations that had been performed showed a pure obstructive ventilatory abnormality – findings were not consistent with a coal dust related condition but rather a condition such as smoking, pulmonary emphysema, non-occupational chronic

bronchitis, and asthma; (3) there was no impairment in oxygen transfer as Frasure did not become hypoxic with exercise. Believed that Frasure was quite functionally disabled because of his severe obstructive lung disease due to smoking; however, the impairment and disability was unrelated to the inhalation of coal mine dust.

24. DX 62 — Anderson, Board-certified internal medicine and pulmonary disease — 9-19-94

Opinion: Noted that the majority of x-ray readings were negative for pneumoconiosis. Opined with a high degree of medical certainty that Frasure did not have the respiratory capacity to perform hard manual labor. His impairment was due to smoking, not coal dust. He had the typical bronchitis and emphysema of long-term smokers.

25. DX 92 — Vuskovich — 9-25-95

Opinion: Recorded a smoking history of up to four packs per day. Started smoking as a child and quit in 1985. He would have a 100 + pack per year smoking history. It is possible to distinguish with a degree of medical certainty between pulmonary disability caused by smoking and that caused by coal exposure. Facts in this record show that Frasure has chronic obstructive pulmonary disease secondary to cigarette abuse. Significant smoking history plus evidence of moderate to severe obstructive impairment supports this conclusion. Frasure did not have any pulmonary disability or impairment arising from his occupation as a coal miner.

26. DX 93 — Fino, B-reader, Board-certified in Internal Medicine with subspecialty in pulmonary disease — 9-27-95

Opinion: Newly submitted evidence did not cause Fino to change his opinion in previous report. Majority of chest x-ray readings are negative for pneumo. Spirometric evaluationss show a pure obstructive ventilatory abnormality. That is based on a reduction in the FEV1/FVC ratio. Obstructive ventilatory abnormality has occurred in the absence of any restrictive defect. Frasure does not have a restrictive defect, and shows a pure obstructive defect. Also, the obstruction shows involvement in the small airways. Large airway flow is measured by the FEV1 and FEV1/FVC ratio. Small airway flow is measured by the FEF 25-75. On a proportional basis, the small airway flow is more reduced than the large airway flow. This type of finding is not consistent with coal dust related condition but with smoking, pulmonary emphysema, non-occupational chronic bronchitis, and asthma. Frasure has improvement following administration of bronchodilators. This implies that the cause of the obstruction is not fixed and permanent – pneumoconiosis is obviously a fixed condition.

27. DX 98 — Branscomb, B-reader, Board-certified in Internal Medicine — 10-6-95

Opinion: Opined that restrictive impairment is not present for the following reason: When obstruction is severe and much air is trapped into a hyperinflated chest there is insufficient room for a full vital capacity volume. Thus, a low FVC value is a reflection of obstruction, not restriction, in this situation. The hyperinflated condition, as opposed to the small shrunken lung of restricted disease, was confirmed by all x-rays. Diagnosed severe chronic obstructive pulmonary disease secondary to smoking in a susceptible host. Findings are those of chronic airways obstruction with a great deal of reversibility. Findings are typical of this disorder as it occurs in general population unrelated to coal mining. Would be the same had Frasure never been involved in coal mining.

Discussion

Material Change in Condition

Because this claim involves a duplicate claim, it is first necessary to evaluate whether the Claimant can establish a material change in condition since the denial of his prior claim. As explained earlier, to demonstrate that a change in condition has occurred since the denial of his prior claim, Frasure must prove, based on evidence developed since May 2001, the threshold issue of existence of pneumoconiosis pursuant to 20 C.F.R. § 718.202. For the reasons set forth below, I conclude that Claimant has failed to achieve this threshold showing. In addition, I note that many of the physicians consulted for their medical opinions in this case tended to conflate a discussion of the existence of pneumoconiosis with a discussion of etiology and total disability. While these are, of course, separate inquiries pursuant to 20 C.F.R. §§ 718.203, 718.204, that are made once the existence of pneumoconiosis has been established, I will nevertheless address them insofar as they seem central to the threshold issue of whether pneumoconiosis existed.

Existence of Pneumoconiosis

Pneumoconiosis is defined by the Regulations as "chronic dust disease of the lung and its sequelae, including respiratory and pulmonary impairments, arising out of coal mine employment." 20 C.F.R. § 718.201. The definition is not confined to 'coal workers' pneumoconiosis,' but also includes other diseases arising out of coal mine employment, such as anthracosilicosis, anthracosis, anthrosilicosis, massive pulmonary fibrosis, progressive massive fibrosis, silicosis, or silicotuberculosis. 20 C.F.R. § 718.201.

This broad definition "effectively allows for the compensation of miners suffering from a variety of respiratory problems that may bear a relationship to their employment in the coal mines." ***Robinson v. Pickands Mather & Co./Leslie Coal Co. & Director, OWCP,*** 14 B.L.R. 2-68, 2-78 (CA4 1990), 914 4th Cir. 1990), citing ***Rose v. Clinchfield Coal Co.,*** 614 F.2d 936, 938 (4th Cir. 1980). Thus, asthma, asthmatic bronchitis or emphysema may fall under the regulatory definition of pneumoconiosis if they are related to coal dust exposure. ***Robinson v. Director, OWCP,*** 3 B.L.R. 1-798.7 (1981); ***Tokarcik v. Consolidation Coal Co.,*** 6 B.L.R. 1-666 (1983)(chronic bronchitis secondary to coal dust exposure equivalent to CWP); ***Heavilin v. Consolidation Coal Co.,*** 6 B.L.R. 1-1209 (B.R.B. 1984)(emphysema held compensable under the Act). Likewise, chronic obstructive pulmonary disease (COPD) may be encompassed within the legal definition of pneumoconiosis. ***Warth v. Southern Ohio Coal Co.,*** 60 F.3d 173 (4th Cir. 1995)(COPD refers to three disease processes – chronic bronchitis, emphysema and asthma – that are all characterized by airway dysfunction).

The claimant has the burden of proving the existence of pneumoconiosis. The Regulations provide the means of establishing the existence of pneumoconiosis by one (1) of the following methods: (1) chest x-ray evidence; (2) autopsy or biopsy; (3) by operation of presumption; or (4) by "other relevant evidence." 20 C.F.R. 718.202(a)(1-4).

a. X-Ray Evidence

Section 718.202(a)(1) provides for a finding of the existence of pneumoconiosis with positive chest x-ray evidence, and that "where two or more x-rays are in conflict, in evaluating such x-ray reports, consideration shall be given to the radiographic qualifications of the physicians interpreting such x-rays." 20 C.F.R. § 718.202(a)(1). Positive x-rays may form the basis of a finding of the existence of pneumoconiosis; however, they must be considered in light of all the relevant evidence. I am not to blindly defer to the numerical superiority of x-ray evidence, ***Adkins v. Director, OWCP,*** 958 F.2d 49, 52 (4th Cir. 1992); ***Woodward v. Director, OWCP,*** 991 F.2d 314 (6th Cir. 1993); ***Sahara Coal Co. v. Fitts,*** 39 F.3d 781 (7th Cir. 1994); ***Wilt v. Wolverine Mining Co.,*** 14 B.L.R. 1-70 (1990), although it is within my discretion to do so. ***Edminston v. F & R Coal Co.,*** 14 B.L.R. 1-65 (1990).

Box 2B(c) of the standard x-ray form indicates the quantity of opacities in the lung and therefore, the presence or absence of pneumoconiosis. The more opacities noted in the lung, the more advanced the disease; and there are four (4) categories to which a physician may choose:

> **0** = small opacities absent or less than in category 1;
> **1** = small opacities definitely present, but few in number;
> **2** = small opacities numerous, but normal lung markings still visible;
> **3** = small opacities very numerous and normal lung markings are usually partly or totally obscured.[9]

If no categories are chosen, then the x-ray report is not classified according to the standards adopted by the regulations and cannot, therefore, support a finding of pneumoconiosis. Likewise, an x-ray which is interpreted as Category 0 (–/0, 0/0, or 0/1) demonstrates, at most, only a negligible presence of the disease and will not support a finding of pneumoconiosis under the Act or regulations.

If the physician determines that the study is Category 1 (1/0, 1/1 or 1/2), Category 2 (2/1, 2/2 or 2/3) or Category 3 (3/2, 3/3 or 3/+), then there is a definite presence of opacities in the lung and the x-ray report may be used as evidence of pneumoconiosis. An interpretation of 1/0 is the minimum reading under the regulations which will support a finding of pneumoconiosis. A 1/0 reading indicates that the physician has determined that the x-ray is Category 1, but he/she seriously considered Category 0. As for another example, a reading of 2/2 indicates that the physician determined that the x-ray was Category 2 and Category 2 was the only other category seriously considered by the physician.

The new x-ray evidence in this case consists of three x-ray re-readings by Dr. Poulos (DX 162) and one original radiology report prepared by Dr. Rice. (DX 155). Two of the x-ray re-readings by Dr. Poulos are of x-rays allegedly taken at Highlands Regional Medical Center on January 5, 2001 and May 4, 2001. I note that, while copious medical records from Frasure's hospitalizations at Highlands Regional Medical Center are in evidence (DX 134, 154), these records remarkably do not include the original x-ray reports from January 5, 2001, and May 4, 2001. The third x-ray re-reading is of an x-ray taken at Central Baptist Hospital on August 28,

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[9] 20 C.F.R. §§ 718.108 Chest Roentgenograms (x-rays).

2000. The original radiology report, prepared by Dr. Rice at Central Baptist hospital, is in evidence. (DX 155).

Dr. Poulos, who is a B-reader and Board-certified Radiologist, concluded that the x-ray allegedly taken at Highlands Regional Medical Center on May 4, 2001, demonstrated no evidence of pneumoconiosis. He also noted that the film quality was "Grade 3" and overexposed. He determined that the x-ray allegedly taken at Highlands Regional Medical Center on January 5, 2001 was unreadable due to over-exposure. Finally, Dr. Poulos found that the x-ray taken at Central Baptist Hospital on August 28, 2000 was also unreadable due to over-exposure. Dr. Rice, the physician who prepared the original radiology report of the August 28, 2000 x-ray, interpreted: "(1) Mild cardiomegaly, compensated; (2) Chronic pulmonary changes; (3) Groshong catheter is well positioned. There is no pneumothorax."

I conclude that these x-rays fail to prove the existence of pneumoconiosis. Dr. Poulos, who is well-qualified as a B-reader and Board-certified Radiologist, interpreted one of the x-rays as negative and two of the x-rays as unreadable. Dr. Rice, whose qualifications were not made available, also failed to detect pneumoconiosis in the radiology report he prepared. Claimant has not submitted any positive x-ray readings since the prior denial of benefits, and has therefore failed to prove the existence of pneumoconiosis through x-ray evidence.

b. Autopsy or Biopsy Evidence

As there is no autopsy or biopsy evidence in the record, Section 718.202(a)(2) is not applicable.

c. Presumptions

Under Section 718.202(a)(3), the existence of pneumoconiosis may be established through the application of the presumptions described in Sections 718.304, 718.305 or 718.306. Section 718.304 requires x-ray, biopsy or equivalent evidence of complicated pneumoconiosis which is not present in this case. The rebuttable presumption of Section 718.305 is not available to the Claimant because he filed his application after January 1, 1982. Section 718.306 is only applicable in the case of a deceased miner who died on or before March 1, 1978 and who was employed twenty-five (25) or more years prior to June 30, 1971. This is not the case here either.

d. Other Relevant Evidence

A determination of the existence of pneumoconiosis may be made if a physician, exercising sound medical judgment, notwithstanding negative x-ray evidence, finds that the miner suffers from pneumoconiosis as defined in § 718.201. 20 CFR §§ 718.202(a)(4) (2003). Thus, even if the x-ray evidence is negative, medical opinions may establish the existence of pneumoconiosis. ***Taylor v. Director, OWCP***, 9 B.L.R. 1-22 (1986). The medical opinions must be reasoned and supported by documented, objective medical evidence such as blood gas studies, electrocardiograms, pulmonary function studies, physical performance tests, physical examination, and medical and work histories. 20 CFR § 718.202(a)(4) (2003).

Specifically, a "documented" opinion is one that sets forth the clinical findings, observations, facts, and other data upon which the physician based the diagnosis. ***Fields v. Island Creek Coal Co.***, 10 B.L.R. 1-19, 1-22 (1987). An opinion may be adequately

documented if it is based on items such as a physical examination, symptoms, and the patient's work and social histories. ***Hoffman v. B&G Construction Co.***, 8 B.L.R. 1-65, 1-66 (1985); ***Hess v. Clinchfield Coal Co.***, 7 B.L.R. 1-295, 1-296 (1984); ***Justus v. Director, OWCP***, 6 B.L.R. 1-1127, 1-1129 (1984). A "reasoned" opinion is one in which the judge finds the underlying documentation and data adequate to support the physician's conclusions. ***Fields,*** above. Whether a medical report is sufficiently documented and reasoned is for the judge to decide as the finder-of-fact; an unreasoned or undocumented opinion may be given little or no weight. ***Clark v. Karst-Robbins Coal Co.***, 12 B.L.R. 1-149, 1-155 (1989) (en banc). An unsupported medical conclusion is not a reasoned diagnosis. ***Fuller v. Gibraltar Corp.***, 6 B.L.R. 1-1291, 1-1294 (1984). *See also* ***Phillips v. Director, OWCP***, 768 F.2d 982 (8th Cir. 1985); ***Smith v. Eastern Coal Co.***, 6 B.L.R. 1-1130 (1984); ***Duke v. Director, OWCP***, 6 B.L.R. 1-673 (1983)(a report is properly discredited where the physician does not explain how underlying documentation supports his or her diagnosis); ***Waxman v. Pittsburgh & Midway Coal Co.***, 4 B.L.R. 1-601 (1982). Also, a physician's report may be rejected where the basis for the physician's opinion cannot be determined. ***Cosaltar v. Mathies Coal Co.***, 6 B.L.R. 1-1182 (1984).

Additionally, the qualifications of the physicians are relevant in assessing the respective probative values to which their opinions are entitled. ***Burns v. Director, OWCP***, 7 B.L.R. 1-597, 1-599 (1984). I note that the new treating physician regulation does not apply retroactively: "With respect to treating physicians' opinion developed and submitted before the effective date of the final rule, the judicial precedent summarized in the Department's initial notice of proposed rule-making continues to apply. *See* 62 Fed. Reg. 3342 (January 22, 1997). These decisions recognize that special weight may be afforded the opinion of a miner's treating physician based on the physician's opportunity to observe the miner over a period of time.

Regulations Implementing the Federal Coal Mine Health and Safety Act of 1969, 65 Fed. Reg. 79,334 (Dec. 20, 2000).

With regard to medical opinions, Claimant relies primarily on the deposition testimony of his treating physician, Dr. Ayesha Sikder, taken August 2, 2001. I conclude that Sikder's testimony is not well-reasoned in several respects. The basis for her opinion that Frasure suffered from coal workers' pneumoconiosis often appears equivocal and vague. Finally, her opinion is not well-documented. In attesting to the presence of pneumoconiosis, Sikder states that she relied on Frasure's fourteen-year history of coal mine employment, his pulmonary function study, and chest x-rays. (DX 134:14-15). Sikder had an accurate understanding of the length of Frasure's coal mine employment as being fourteen years in that the parties have stipulated to this length. Length of coal mine employment alone, however, is insufficient to prove the existence of pneumoconiosis. Sikder's explanation of the other two factors that led her to deduce the existence of pneumoconiosis, the pulmonary function study and chest x-rays, is not well-reasoned nor is it well-documented.

First, Sikder based her opinion that Frasure suffered from pneumoconiosis in large part on the results of a pulmonary function study. However, the Board has specifically held that pulmonary function studies are not diagnostic of the presence or absence of pneumoconiosis. ***Burke v. Director, OWCP***, 3 B.L.R. 1-410 (1981). That notwithstanding, I will address Sikder's conclusions regarding the pulmonary function study insofar as she alleges very few other reasons for her finding of pneumoconiosis. In her deposition, Sikder observed that

Frasure's pulmonary function study revealed severe obstructive airway disease with concomitant restrictive lung disease. (DX 134:8). By concomitant, Sikder meant that he had "reduced FEV1, which is suggestive of airway disease, but he also [had] reduced FVC, which is suggestive of restrictive airway disease." (DX 134:9). Specifically, she reported that Frasure's FVC was 1.7 liters, which is 40 percent of predicted, and his FEV1 was 0.75, which is 23 percent predicted. (DX 134:9). Sikder testified that, according to the American Thoracic Society classifications, usually less than 40 percent predicted is severe disease. (DX 134:9). Thus, she opined, that 23 percent of predicted represents severe obstructive disease and is suggestive of end stage lung disease (i.e. lung disease that is so far advanced that there will be no meaningful recovery). (DX 134:9). She added that FVC being 40 percent of predicted also suggests restrictive defect. (DX 134:9). Moreover, Sikder considered the test to be valid in that it was very reproducible (i.e. four trials were done that yielded nearly identical values) and the flow volume group was classic for obstructive airway disease. (DX 134:10).

Next, in asserting that Frasure's chest x-rays demonstrated the existence of pneumoconiosis, Sikder testified that she "looked at Frasure's chest x-rays on several occasions and noticed chronic infiltrates, which are suggestive of coal dust exposure. (DX 134:15). She failed, however, to identify or provide these x-rays at her deposition. At another point in her deposition, Sikder stated that she looked at "probably 40 x-rays." (DX 134:16). Again, she failed to identify or provide these forty x-rays. While a copy of the numerous x-rays submitted in the modification claim was attached to her deposition, it is unclear which, if any, of these x-rays were relied upon by Sikder. As noted above, the Board in ***Fields*** has held that a "documented" opinion is one that sets forth the clinical findings, observations, facts, and other data upon which the physician based the diagnosis. As also noted above, the Board in ***Cosaltar*** has held that a physician's report may be rejected where the basis for the physician's opinion cannot be determined. Because she failed to set forth the specific x-rays upon which her conclusions were based, I find Sikder's opinion not well-documented. Considering that much of her opinion seems to rely on chest x-ray evidence, the fact that she fails to clearly identify the chest x-rays to which she is referring is especially egregious.

In testifying that Frasure's respiratory disease was caused by his coal mine employment, Dr. Sikder claims to have given due consideration to Frasure's smoking history. Indeed, she has an accurate understanding of Frasure's smoking history in that she recorded a smoking history of 50-pack years, the miner having quit in 1985. However, when specifically questioned as to whether coal mine dust exposure caused Frasure's lung disease, her reply was not responsive. (DX 134: 17). She testified that both smoking and coal dust can cause lung disease and then simply reiterated her position that based on his chest x-ray findings and pulmonary function study, Frasure "clearly had black lung disease." (DX 134:17). In her opinion, Frasure had emphysema, which was caused by both coal dust and tobacco. (DX 134:17). Sikder also stated that chronic obstructive pulmonary disease (COPD) is usually due to cigarette smoke but that coal dust can also cause COPD. (DX 134:18). She further stated that Frasure had COPD. (DX 134:18). She failed to provide her reasoning for this conclusion. (DX 134:17). At this time, she failed to make any further connections or assertions regarding Frasure's COPD. (DX 134:18). Again, an unsupported medical conclusion is not a reasoned diagnosis. *See* ***Fuller.***

In a similar vein, Sikder was also presented with a summary of x-ray interpretations that had been submitted in the prior decisions regarding this claim. (DX 134:21). The summary included readings by B-readers and Board-certified radiologists. (DX 134:22). Sikder was then questioned as to whether her opinion that Frasure's COPD was caused at least in part by coal dust exposure would change if the weight of the x-ray interpretations was negative for classic medical pneumoconiosis. (DX 134:22). She stated that her opinion would not change because often chest x-rays are negative in patients with coal dust exposure and this later becomes revealed when lung biopsies are done. (DX 134:22). While the generality may be true that there are cases where biopsies reveal the existence of pneumoconiosis even when chest x-rays are negative, no biopsy evidence has been submitted in this case. The Board has held that a medical opinion based upon generalities, rather than specifically focusing upon the miner's condition, may be rejected. *See* ***Knizer v. Bethlehem Mines Corp.***, 8 B.L.R. 1-5 (1985). By alleging a generality, rather than focusing on the specific evidence that has been submitted in this case, Sikder again fails to provide a useful opinion with regard to the existence of pneumoconiosis in this case. The x-ray evidence does not support Dr. Sikder's conclusions.

As noted above, special weight *may* be afforded to the opinion of a miner's treating physician based on the physician's opportunity to observe the miner over a period of time. (*emphasis added*). Although Dr. Sikder was Frasure's treating physician at the time she gave her deposition, I find the reasoning and documentation throughout her opinion grossly inadequate. Therefore, not only do I decline to accord greater weight to her opinion as the treating physician, I also find, for the reasons stated above, that her opinion should be accorded minimal weight. I also note that, while Dr. Sikder is Board-certified in Internal Medicine and completed a fellowship in pulmonary medicine, these credentials do not offset the lack of reasoning and documentation throughout her opinion such that I can grant any significant weight to her opinion.

In addition to the deposition testimony of Dr. Sikder, Claimant also submitted copious medical records from Frasure's multiple hospitalizations at both Highlands Regional Medical Center and Central Baptist Hospital. While many of these hospitalizations were due to Claimant's respiratory problems, they are not probative with regard to whether Frasure suffered specifically from coal workers' pneumoconiosis. Many of the hospital records state that Claimant suffered from coal workers' pneumoconiosis; however, these statements appear conclusory as the records fail to articulate how this determination was reached.[10] As noted above, the Board in ***Fuller*** has held that an unsupported medical conclusion is not a reasoned diagnosis. *See also* ***Phillips v. Director, OWCP***, 768 F.2d 1982 (8th Cir. 1985); ***Smith v. Eastern Coal Co.***, 6 B.L.R. 1-1130 (1984); ***Duke v. Director, OWCP***, 6 B.L.R. 1-673 (1983)(a report is properly discredited where the physician does not explain how underlying documentation supports his or her diagnosis); ***Waxman v. Pittsburgh & Midway Coal Co.***, 4 B.L.R. 1-601 (1982). For this reason, the numerous hospital records, which simply include the conclusory statement that Frasure suffered from coal workers' pneumoconiosis, are not persuasive on the issue of the existence of pneumoconiosis.

[10] In this regard, I note that Dr. Sikder authored 20 of the 39 hospital records submitted in this case. Thus, many of the records diagnosing coal workers' pneumoconiosis were written by a physician whose opinion I have already accorded little credence.

In his medical report, Dr. Broudy, who is a B-reader and Board-certified in Internal Medicine and Pulmonary Medicine, criticized Dr. Sikder's assertion that pneumoconiosis existed in this case, observing that Sikder attributed the miner's impairment to his coal dust exposure even though the x-rays were largely read as negative for pneumoconiosis. (EX 1). Broudy looked not only at new x-ray readings submitted since the denial of Frasure's claim but at all x-rays that have been submitted in this case. Thus, his report is instructive regarding Frasure has established a change in condition (i.e. the existence of pneumoconiosis) based on new x-ray evidence since the prior denial. However, Broudy mistakenly noted in his report that Dr. Poulos read x-rays dated August 28, 2000, January 5, 2001, and May 4, 2001 as negative for pneumoconiosis. In fact, Dr. Poulos read only the May 4, 2001 x-ray as negative; he determined that the x-rays dated August 28, 2000 and January 5, 2001 were unreadable.

Broudy also determined that there were sufficient facts in the record to demonstrate that the miner's disability resulted from chronic obstructive pulmonary disease due to cigarette smoking rather than coal dust. (EX 6). First, he noted Frasure's significant history of smoking at a rate of a pack per day for fifty years. (EX 6:10). He testified that this smoking history is certainly sufficient to cause chronic obstructive airways disease. (EX 6:10). In this regard, I note that Broudy's opinion is not particularly well-reasoned. Just as Dr. Sikder failed to explain why she opined that coal dust rather than smoking caused Frasure's impairment, Dr. Broudy failed to explain why he opined that smoking rather than coal dust caused Frasure's impairment.

On the other hand, Broudy noted that Frasure had the typical impairment associated with smoking: chronic airways obstruction. He distinguished this type of defect from a restrictive defect, which he explained one might expect to see with disabling impairment due to pneumoconiosis. (EX 6:10). Whether Frasure suffered from both a restrictive and obstructive defect or merely an obstructive defect is clearly a source of contention. However, regardless of the accurate answer to this question, I note again that pulmonary function studies are not diagnostic of the presence or absence of pneumoconiosis. ***Burke v. Director, OWCP***, 3 B.L.R. 1-410 (1981). Thus, I find Broudy's opinion unavailing in this regard as well.

Finally, Broudy testified that, while not impossible, it was extremely rare for simple pneumoconiosis to cause a severe impairment of that type that Frasure had. (EX 6: 21). He articulated that "[a] vast majority of cases of severe respiratory impairment due to pneumoconiosis occur in those patients who have complicated pneumoconiosis or progressive massive fibrosis." (EX 6:20-21). Such was not the case here.

In sum, I find Broudy's medical opinion more persuasive than Sikder's medical opinion. Although Broudy's opinion was not well-documented with regard to x-ray evidence, not well-reasoned with regard to the impact of Frasure's smoking history, and not dispositive with regard to pulmonary function testing, his opinion is consistent with the findings established by the x-ray evidence, and he at least provided one reasoned conclusion that normally only complicated pneumoconiosis causes the type of severe impairment that Frasure had. Broudy is also somewhat more qualified than Sikder in that he is a B-reader and is Board-certified in Pulmonary Medicine as opposed to Sikder who only completed a fellowship in Pulmonary Medicine.

Dr. Dahhan, who is a B-reader and Board-certified in Internal Medicine and Pulmonary Medicine, also testified that Frasure had an obstructive defect, which he opined would be inconsistent with coal workers' pneumoconiosis. (EX 4:9). He asserted that Sikder based her finding of restrictive ventilatory defect on invalid functions. (EX 4:9). In his medical report, Dr. Dahhan disagreed with Sikder that Frasure's reduction in FVC indicated a restrictive ventilatory defect. (EX 2). Dahhan explained that when a patient has lost so much long function, regardless of the cause, that patient will experience a reduction in FVC. Thus, many patients with advanced emphysema, such as Frasure, demonstrate a reduction in FVC as well as a marked reduction in FEV1. (EX 2). Dahhan stated that overall the pulmonary function studies performed on Frasure were indicative of a severe obstructive ventilatory defect with no evidence of a restrictive ventilatory defect. (EX 2). Again, while there is clear disagreement as to whether pulmonary function studies show that Frasure suffered from both a restrictive and obstructive defect or merely an obstructive defect, this dispute begs the question of whether pneumoconiosis existed in this case.

In his deposition, Dahhan also noted that Frasure produced various responses to the pulmonary function studies depending on the administration of medication. (EX 4:5-6). Stated differently, Frasure's defect was not "completely fixed," but rather "wax[ed] and wan[ed]" depending on whether medication was administered. (EX 4:6). Presumably, therefore, Frasure did not suffer from pneumoconiosis, which is an irreversible, progressive disease. *See* ***Woodward v. Director, OWCP***, 991 F.2d 314, 320 (6th Cir. 1993). In this regard, I accept Dahhan's conclusion as a well-reasoned tending to show the absence of pneumoconiosis.

Finally, Dahhan noted that Frasure's obstructive defect resulted from chronic bronchitis and emphysema. Dahhan articulated that simple coal workers' pneumoconiosis causes a form of emphysema known as focal emphysema. Focal emphysema involves a dilation of the alveolar sacs in the area surrounding the coal macule. It is not associated with any destruction of the alveolar structure or loss in the mechanics of the respiratory system as Frasure experienced. Thus, Dahhan deduced that Frasure suffered from cigarette induced centriacinar and panacinar emphysema, rather than focal emphysema. I also accept this conclusion as a better reasoned, argument tending to show that Frasure's impairment was caused by cigarette smoking rather than coal dust exposure.

In sum, I find Dahhan's medical opinion more persuasive than the opinions of both Broudy and Sikder. Although Dahhan made the same non-dispositive argument regarding obstructive impairment in the context of whether pneumoconiosis existed, he also provided two compelling arguments regarding irreversibility of the disease and focal emphysema. In addition, I note that Dahhan's qualifications match those of Broudy in that he too is a B-reader and Board-certified in Pulmonary Medicine. In this regard, Dahhan is also more qualified than Sikder.

In his deposition, Dr. Fino, who is a B-reader and Board-certified in Internal Medicine and Pulmonary Medicine, asserted that there were sufficient facts in the record to demonstrate that Frasure had pulmonary disability caused by cigarette smoking rather than coal dust. (EX 5:11). These facts included the pattern of abnormality in the lung function studies and the type of changes in the blood gas system. (EX 5:9).

With regard to the blood gas studies, Fino specifically observed that the resting blood gases in the arterial blood gas studies performed prior to Frasure's many hospitalizations were generally normal. Although there were a couple of abnormal blood gases prior to 1993, any resting hypoxemia experienced by Frasure would disappear and reappear. (EX 5:6). For example, blood gases that showed hypoxemia in 1985 no longer showed hypoxemia in 1993. Fino explained that this phenomenon is significant because if one has a coal dust related disease causing hypoxia then the hypoxia is present at all times (i.e. it does not disappear and reappear). (EX 5:6). In addition, on two exercise studies in 1993, Frasure did not drop his blood oxygen level with exercise, which again indicates the unlikelihood that coal mine dust contributed to Frasure's respiratory impairment. (EX 5:6). Fino observed that on December 29, 1999, Frasure actually maintained a room air arterial blood gas that was normal with a pO2 of 79. (EX 5:6). I accept Fino's observations in this regard as a well-reasoned opinion tending to show that Frasure did not suffer from pneumoconiosis nor did it contribute to his respiratory impairment.

Fino also remarked that in the year 2000, Frasure had numerous problems that required him to be admitted to the hospital. (EX 5:6). At this time, Frasure's blood gases began to worsen. (EX 5:6). Also at this time, Frasure on several medications, all but one of which were being used to treat obstructive lung disease. (EX 5:7). Fino explained that these medications were being used to treat an obstructive disease that was caused by cigarette smoking because it is not possible to treat an obstructive disease caused by coal dust exposure as this is a permanent and irreversible condition that does not improve with medication. (EX 5:7). *See* ***Woodward*** above. I note that, while the hospital records show that Frasure was taking myriad medications toward the end of his life, there is no evidence that any of these medications improved his condition. Had Frasure responded positively to the medications, then Fino's point regarding the irreversibility of pneumoconiosis would be well taken. However, this was not the case. Therefore, I do not accept this argument as well-reasoned.

Finally, Fino observed that toward the end of his life, Frasure began to experience hypercarbia, a condition in which a patient experiences elevations in his carbon dioxide level. (EX 5:11). Fino explained that one does expect to see hypercarbia in cases where a patient has coal workers' pneumoconiosis, except in cases of severe scarring and fibrosis as is present in complicated pneumoconiosis (and none of the x-rays in this case were read as showing complicated pneumoconiosis). I accept Fino's observations in this regard as a well-reasoned opinion tending to show that Frasure did not suffer from pneumoconiosis

In sum, I find Fino's medical opinion to be equally as compelling as Dahhan's opinion. I accepted all but one of Fino's observations as well-reasoned. I also that Fino's qualifications match those of Broudy and Dahhan and superior to those of Sikder.

Claimant has clearly failed to meet his burden of proof in attempting to establish the existence of pneumoconiosis through medical opinion. Claimant relied primarily on Dr. Sikder's testimony, which is grossly inadequate with regard to documentation and reasoning. While I note that Sikder is Board-certified in Internal Medicine, completed a pulmonary fellowship, and acted as Claimant's treating physician, her opinion must be discounted, as her opinion is flawed. Moreover, the hospital records submitted by Claimant contain conclusory diagnoses and provide

no insight or reasoning as to the presence or absence of pneumoconiosis. Claimant failed to put forth sufficient evidence to satisfy his burden of proof.

The medical opinions of three physicians, Broudy, Dahhan, and Fino, all concluded the absence of pneumoconiosis. All three of these physicians are B-readers, Board-certified in Internal Medicine, and Board-certified in Pulmonary Medicine. Given these qualifications, their opinions are entitled to great weight. As articulated above, I did not accept all of the arguments expressed in these medical opinions but found certain arguments to be particularly persuasive. This is more than can be said for the Claimant, who failed to meet his burden of proof on the issue of whether pneumoconiosis existed. ***Oggero****, supra*.

The totality of the evidence submitted since the prior denial of benefits shows that pneumoconiosis is not established by x-ray. 20 C.F.R. § 718.202(a)(1). Moreover, the Claimant has not provided a documented or reasoned report from a physician, who, exercising sound medical judgment, notwithstanding a negative x-ray, finds that the miner suffered from pneumoconiosis. 20 C.F.R. § 718.202(a)(4). After a review of all the evidence, I find that pneumoconiosis has not been established under 20 C.F.R. § 718.202(a)(1)-(4). Therefore, I find that Claimant has failed to prove a material change in condition.

Mistake in Determination of Fact

Although I find that Claimant has failed to establish a change in condition, the record in this case is re-opened to admit the evidence submitted by Claimant and Employer to determine if a mistake of fact was made in determining that Claimant did not suffer from coal workers' pneumoconiosis. In his November 7, 1997 Decision and Order on Remand Awarding Benefits, the administrative law judge (ALJ) found that Claimant failed to establish the existence of pneumoconiosis through x-ray evidence but was able to prove its existence through medical opinion. Accordingly, in his April 25, 2000 Decision and Order on Remand Denying Benefits, the ALJ did not reconsider the x-ray evidence but focused exclusively on the weight of the medical opinion evidence in concluding that Frasure did not suffer from coal workers' pneumoconiosis. (DX 116).[11] Notwithstanding that the ALJ did not reconsider the x-ray evidence in his most recent decision, I will, in the interest justice, consider the x-ray evidence and the relevant medical opinions to determine whether there was a mistake in determination of fact regarding whether Frasure suffered from coal workers' pneumoconiosis.

A review of the radiographic interpretation evidence reveals a conflict in opinion as to whether the Claimant suffers from coal workers' pneumoconiosis. In such cases, numerous guidelines exist for evaluating the diverse interpretations. First, the actual number of interpretations favorable and unfavorable may be a factor. ***Wilt v. Wolverine Mining Company,*** 14 B.L.R. 1-70 (1990). At the same time, mechanical reliance on numerical superiority is not appropriate. ***Adkins v. Director, OWCP,*** 958 F.2d 49 (4th Cir. 1992). Second, consideration may be given to the evaluating physicians' qualifications and training. ***Dixon v. North Camp Coal,*** 8 B.L.R. 1-344 (1985); ***Melink v. Consolidation Coal Company,*** 16 B.L.R. 1-31 (1991). The interpretations from the doctors with the greater expertise may be accorded more evidentiary weight. ***Taylor v. Director, OWCP,*** 10 BRBS 449, BRB No. 77-610 BLA (1979). The qualifications of the doctor who provided the most recent evaluation may also bear on the

[11] As stated previously, the ALJ, therefore, did not the issue of total disability. (DX 116).

evidentiary weight of the study. ***McMath v. Director, OWCP,*** 12 B.L.R. 1-6 (1988). Finally, when faced with multiple interpretations of numerous x-rays, an administrative law judge should first evaluate the conflicting interpretations on one (1) x-ray to determine whether that particular x-ray is negative or positive. Then, the administrative law judge resolves the conflict between the x-rays in context to determine whether pneumoconiosis is present. ***Copley v. Arch of West Virginia, Inc.,*** Case No. 93-1940 (4th Cir. June 21, 1994)(unpublished).

I will first provide a summary of the interpretations regarding the various x-rays taken in 1985. With regard to the March 11, 1985 x-ray, Drs. Marshall and Brandon, who are both B-readers and Board-certified, read it as positive for pneumoconiosis. Accordingly, this x-ray will be considered positive. With regard to the June 19, 1985 x-ray, Dr. deGuzman, who is neither a B-reader nor Board-certified, read it as positive for pneumoconiosis. Accordingly, this x-ray will be considered positive. With regard to the June 20, 1985 x-ray, Dr. Ameji, who is neither a B-reader nor Board-certified, read it as positive for pneumoconiosis. Accordingly, this x-ray will be considered positive. With regard to the August 1, 1985 x-ray, Dr. Lagada, who is neither a B-reader nor Board-certified, read it as positive for pneumoconiosis. Accordingly, this x-ray will be considered positive. With regard to the August 20, 1985 x-ray, Drs. Felson, Wiot, and Spitz, who are all B-readers and Board-certified, read it as negative for pneumoconiosis. Accordingly, this x-ray will be considered negative. With regard to the October 1, 1985 x-ray, Dr. Quillin, who is a B-reader and Board-certified, read it as negative for pneumoconiosis. Dr. Broudy, who is a B-reader, also read it as negative for pneumoconiosis. Accordingly, this x-ray will be considered negative. With regard to the October 15, 1985 x-ray, Dr. Bangudi, who is neither a B-reader nor Board-certified, read it as positive for pneumoconiosis. Accordingly, this x-ray will be considered positive. With regard to the October 21, 1985 x-ray, Dr. El-Amin, who is neither a B-reader nor Board-certified, read it as positive for pneumoconiosis. Accordingly, this x-ray will be considered positive.

Next, I will provide a summary of the interpretations regarding the various x-rays taken in 1993. With regard to the June 28, 1993 x-ray, Drs. Lin and Sundaram, who are neither B-readers nor Board-certified, read it as positive for pneumoconiosis. Dr. Baker, who is a B-reader, read it as positive for pneumoconiosis. Drs. Bassali and Marshall, who are B-readers and Board-certified, also read it as positive for pneumoconiosis. Dr. Broudy, who is a B-reader, read it as negative for pneumoconiosis. Drs. Jarboe, Sargent, Dineen, and Barrett, all of whom are B-readers and Board-certified, read it as negative for pneumoconiosis. Essentially, there are five positive interpretations and five negative interpretations; however, two of the physicians who read the x-ray as positive are neither B-readers nor Board-certified. Therefore, this x-ray will be considered negative. With regard to the August 2, 1993 x-ray, Dr. Sargent, who is a B-reader and Board-certified, read it as negative for pneumoconiosis. Dr. Marshall, who is a B-reader and Board-certified, read as positive for pneumoconiosis. As the equally qualified B/BCR readers found pneumoconiosis to be both present and absent, I find that the readings of this x-ray are in equipoise as to the existence of pneumoconiosis. With regard to the September 25, 1993 x-ray, Drs. Marshall and Brandon, who are B-readers and Board-certified, read it as positive for pneumoconiosis. Dr. Baker, who is a B-reader, read it as positive for pneumoconiosis. Drs. Broudy and Dahhan, who are B-readers, read it as negative for pneumoconiosis. Drs. Sargent, Jarboe, Barrett and Dineen, who are B-readers and Board-certified, read it as negative for pneumoconiosis. Essentially, there are three positive interpretations and six negative

interpretations; moreover, a greater number of physicians who are both B-readers and Board-certified interpreted the x-ray as negative. Accordingly, this x-ray will be considered negative. With regard to the October 12, 1993 x-ray, Dr. Baker, who is a B-reader, read it as positive for pneumoconiosis. Drs. Brandon and Marshall, who are B-readers and Board-certified, read it as positive for pneumoconiosis. Dr. Broudy, who is a B-reader, read it as negative for pneumoconiosis. Drs. Sargent, Binns, Abramowitz, Dineen, and Gogineni, all of whom are B-readers and Board-certified, read it as negative for pneumoconiosis. Essentially, there are three positive interpretations and six negative interpretations; moreover, a greater number of physicians who are both B-readers and Board-certified interpreted the x-ray as negative. Accordingly, this x-ray will be considered negative.

Next, I will provide a summary of the interpretations regarding the x-ray taken in 1994. With regard to the June 28, 1994 x-ray, Drs. Sundaram and Wright, who are neither B-readers and Board-certified, read it as positive for pneumoconiosis. Dr. Baker, who is a B-reader, read it as positive for pneumoconiosis. Drs. Marshall, Brandon, and Bassali, who are B-readers and Board-certified, read it as positive for pneumoconiosis. Drs. Sargent, Abramowitz, Wershba, and Gogineni, who are B-readers and Board-certified, read it as negative for pneumoconiosis. Essentially, there are six positive interpretations and four negative interpretations. Although the number of positive interpretations outweighs the number of negative interpretations, I note that the qualifications of those physicians who interpreted the x-ray as negative are, on the whole, superior to those who read it as positive. Therefore, the interpretations are considered in equipoise.

Next, I will provide a summary of the interpretations regarding the various x-rays taken in 1995. With regard to the January 6, 1995 x-ray, Dr. Myer, who is neither a B-reader nor Board-certified, read it as positive for pneumoconiosis. Dr. Marshall, who is a B-reader and Board-certified, read it as positive for pneumoconiosis. Drs. Sargent, Binns, Abramowitz, and Gogineni, who are B-readers and Board-certified, read it as negative for pneumoconiosis. Essentially, there are two positive interpretations and four negative interpretations; moreover, a greater number of physicians who are both B-readers and Board-certified interpreted the x-ray as negative. Accordingly, this x-ray will be considered negative. With regard to the January 7, 1995 x-ray, Dr. Wright, who is neither a B-reader nor Board-certified, read it as positive for pneumoconiosis. Accordingly, this x-ray will be considered positive. With regard to the February 3, 1995 x-ray, Dr. Broudy, who is a B-reader, read it as negative for pneumoconiosis. Drs. Jarboe, Binns, Sargent, and Wershba, all of whom are B-readers and Board-certified, also read it as negative for pneumoconiosis. Drs. Marshall and Brandon, who are B-readers and Board-certified, read it as positive for pneumoconiosis. Essentially, there are two positive interpretations and five negative interpretations; moreover, a greater number of physicians who are both B-readers and Board-certified interpreted the x-ray as negative. Accordingly, this x-ray will be considered negative. With regard to the June 19, 1995 x-ray, Dr. Myer, who is a B-reader, read it as positive for pneumoconiosis. Accordingly, this x-ray will be considered positive.

Finally, I will provide a summary of the interpretations regarding the most recent x-rays. The August 28, 2000 x-ray and January 5, 2001 x-ray were deemed unreadable by Dr. Poulos, who is a B-reader and Board-certified, due to the fact that the film was over-exposed. With

regard to the May 4, 2001 x-ray, Dr. Poulos, who is a B-reader and Board-certified, read it as negative for pneumoconiosis. Accordingly, this x-ray will be considered negative.

Overall, I have determined that two of the x-rays are in equipoise, eight of the x-rays are positive, eight of the x-rays are negative, and two of the x-rays are unreadable. While I note the extent to which the x-ray evidence appears to be in equipoise, I also observe that a significant number of the x-rays interpreted as positive were done so by physicians who lack both the B-reader qualification and Board certification. Therefore, if not deemed in equipoise, the x-ray evidence seems to prove the absence of pneumoconiosis. Moreover, in this vein, I reiterate that Claimant bears the burden of establishing the existence of pneumoconiosis by a preponderance of the evidence and that evidence in equipoise is insufficient to sustain Claimant's burden. ***Director, OWCP v. Greenwich Colleries, et al***., 114 S. Ct. 2251, *aff'd sub. nom*. ***Greenwich Colleries v. Director, OWCP***, 990 F.2d 730 (3d Cir. 1993). Because the x-ray evidence appears to be in equipoise, or in the alternative, appears to demonstrate the absence of pneumoconiosis, I find that Claimant has failed to establish the existence of pneumoconiosis pursuant to 20 C.F.R. § 718.202(a)(1).

In his April 25, 2000 Decision and Order on Remand Denying Benefits, the Judge Teitler concluded that, on the whole, the physicians who denied the existence of pneumoconiosis in their medical opinions were more well-qualified than those physicians who asserted the existence of pneumoconiosis. This is decidedly true. Drs. Anderson, O'Neill, Broudy, Dahhan, Branscomb, Fino, Vuskovich deteremined that Claimant did not suffer from pneumoconiosis. By contrast, Drs. Bryson, Sundaram, Ameji, deGuzman, Lagada, El-Amin, Bangudi, Wright, Marshall, Myer, Brandon determined that Claimant did suffer from pneumoconiosis. I note that Dr. Mettu found it possible that Claimant suffered from pneumoconiosis but did not provide a decisive opinion on the issue; accordingly, I will disregard his opinion. In addition, I will disregard the opinion of Dr. Vuskovich, who grossly over-estimated Claimant's smoking history.[12]

While it is true that more physicians found the existence of pneumoconiosis than denied its existence, this numerical superiority loses its value upon closer inspection of the opinions themselves. Of the eleven physicians who concluded that Claimant suffered from pneumoconiosis, one was an A-reader (Bangudi), two were Board-certified in radiology and B-readers (Marshall and Brandon), and one was Board-certified in internal medicine and a B-reader (Myer). Of the eight physicians who concluded that Claimant did not suffer from pneumoconiosis, however, one was Board-certified in internal medicine and pulmonary disease (Anderson), one was a B-reader and Board-certified in Internal Medicine (Branscomb), and three were B-readers and Board-certified in internal medicine and pulmonary disease (Broudy, Dahhan, and Fino). In essence, I find no reason to disrupt the findings of the ALJ's April 25, 2000 Decision and Order on Remand Denying Benefits.

SURVIVOR CLAIM

Claim History

[12] Dr. Vuskovich's opinion was disregarded by the ALJ in his April 25, 2000 Decision and Order on Remand Denying Benefits for the same reason.

Claimant, Scott Frasure, died on October 29, 2001. (DX 152). No autopsy was performed. (Tr. 31). His surviving spouse, Ruth Frasure, filed an application for federal black lung benefits on December 18, 2001. (DX 146). On November 1, 2002, the District Director issued a Proposed Decision and Order awarding benefits to Mrs. Frasure. (DX 167). The Employer appealed. (DX 168). On February 21, 2003, the matter was referred to the Office of Administrative Law Judges. (DX 176). The case was subsequently assigned to me on April 4, 2003. The procedural history described above regarding the filing and ultimate disposition of the Employer's Motion to Strike the deposition testimony of Dr. Sikder taken on August 2, 2001 is also applicable here.

A formal hearing was held on August 19, 2001. Claimant Ruth Frasure failed to attend the scheduled hearing. At the hearing, Director's Exhibits Nos. 134, 154, 155, and 162 were admitted into evidence. (Tr. 23, 24). Employer's Exhibits Nos. 1-7 were admitted into evidence. (Tr. 29, 30, 32, 34). No Claimant's Exhibits, ALJ Exhibits, nor Joint Exhibits were admitted into evidence.

Applicable Standards

A surviving spouse is entitled to benefits if the miner died due to pneumoconiosis which arose out of coal mine employment. See 30 U.S.C. § 901; 20 CFR §§ 718.205 and 725.212(a)(3) (2003). In claims filed after January 1, 1982, death will be considered to be due to pneumoconiosis if (1) competent medical evidence establishes that the miner's death was due to pneumoconiosis; (2) pneumoconiosis was a substantially contributing cause or factor leading to the miner's death or the death was caused by complications of pneumoconiosis; or (3) the presumption set forth at 20 CFR § 718.304 applies, i.e., an irrebuttable presumption that death was due to pneumoconiosis where there is medical evidence of complicated pneumoconiosis; but not if (4) the miner's death was caused by a traumatic injury or the principal cause of death was a medical condition not related to pneumoconiosis, unless the evidence establishes that pneumoconiosis was a substantially contributing cause of death. 20 CFR § 718.205(c) (2003). The Sixth Circuit, in which this claim arises, has held that any condition that hastens the miner's death is a substantially contributing cause of death. *Brown v. Rock Creek Mining Corp*., 996 F.2d 812 (6th Cir. 1993). This principle has now been codified in the regulations at 20 CFR § 718.205(c)(5) (2003).

Issues Presented

Several issues that had been previously contested by the Employer were withdrawn at the formal hearing and will be listed below as stipulations. The remaining contested issues are as follows: (1) *Pneumoconiosis*: whether the miner has/had pneumoconiosis as defined by the Act and the regulations; (2) *Causal Relationship*: whether the miner's pneumoconiosis arose out of coal mine employment; (3) *Total Disability*: whether the miner is/was totally disabled; (4) *Causation*: whether the miner's disability or death is/was due to pneumoconiosis; and (5) *Survivor*: whether the claimant is an eligible survivor of a miner.

Stipulations

At the formal hearing, the following stipulations were made: (1) *Timeliness*: that the claim was timely filed; (2) *Miner*: that the person upon whose death or disability the claim is based is a miner; (3) *Post-1969 Employment*: that the miner worked as a miner after December 31, 1969; (4) *Length of Employment*: that the miner worked at least 14 years in or around one or more coal mines; (5) *Responsible Operator*: that the named employer is the Responsible Operator; (6) *Insurance*: that the named employer has secured the payment of benefits (Sec. 423); (7) *Most Recent Period of Cumulative Employment*: that the miner's most recent period of cumulative employment of not less than one year was with the named Responsible Operator; (8) *Additional Issues*: that the designated Responsible Operator contests additional issues as listed in the letter dated January 29, 2003 (#7 thru 18). (Tr. 34-36).

Medical Evidence

The evidence submitted by Claimant in the survivor claim is precisely the same evidence submitted as new evidence in the modification claim. Claimant submitted DX 134, DX 154, DX 155. Likewise, Employer submitted DX 162 and EX 1-6, (Tr. 29, 32), the same evidence submitted as new evidence in the modification claim. Since this evidence has been summarized above, I will not re-summarize it below.

Discussion

Scott Frasure's death certificate states that his death was caused by respiratory failure due to chronic obstructive pulmonary disease. (DX 152). Moreover, both Claimant's physicians and Employer's physicians agree that Frasure's breathing impairment contributed, in some form, to his death. Physicians disagree, however, with regard to whether pneumoconiosis existed in this case and whether pneumoconiosis, specifically, hastened Frasure's death.

While I note that the applicable standards in a survivor claim are markedly different than those in a modification claim, I also recognize that both claims require a threshold showing of the existence of pneumoconiosis. I was not persuaded that the new evidence submitted by Claimant in the modification claim established the existence of pneumoconiosis, and being that the very same evidence is submitted here, I likewise find that Claimant has failed to establish the existence of pneumoconiosis in the survivor claim.

Notwithstanding that Claimant failed to establish the existence of pneumoconiosis, I would still address those aspects of the medical opinions addressing whether pneumoconiosis hastened Frasure's death. However, Dr. Sikder's deposition, the only medical opinion provided by Claimant, was taken approximately three months before Claimant died. Therefore, it does not address the issue of hastening. While the medical opinions submitted by Employer were rendered after Claimant's death, they too do not address the issue of hastening insofar as they are meant to serve as counter-arguments to Sikder's opinion.

Conclusion

In the living miner's claim, Mrs. Frasure has not established a material change in conditions pursuant to 20 C.F.R. § 725.309(d) since the denial of the prior claim on April 20, 2000. Moreover, the record does not establish that Mr. Frasure suffered from pneumoconiosis or that total disability was caused by pneumoconiosis.

In the survivor's claim, the Claimant has failed to establish that her spouse's demise was caused by or hastened by pneumoconiosis. The Claimant had a duty to provide persuasive evidence of entitlement and has failed to do so. ***Oggero v. Director, OWCP***, *supra*.

Attorney's Fe

The award of an attorney's fee is permitted only in cases in which the Claimant is found to be entitled to benefits under the Act. Since benefits are not awarded in this case, the Act prohibits the charging of attorney's fees to the Claimant for services rendered in pursuit of this claim.

ORDER

IT IS ORDERED that the claim for benefits filed by Ruth Frasure is **denied.**

A

DANIEL F. SOLOMON
Administrative Law Judge

Notice of Appeal Rights. Pursuant to 20 C.F.R. §§ 725.481, any party dissatisfied with this Decision and Order may appeal it to the Benefits Review Board within 30 days from the date this decision is filed with the District Director, Office of Workers' Compensation Programs, by filing a notice of appeal with the Benefits Review Board, ATTN: Clerk of the Board, P.O. Box 37601, Washington, D.C. 20013-7601. See 20 C.F.R. §§§§ 725.478 and 725.479. A copy of a notice of appeal must also be served on Donald S. Shire, Esquire, Associate Solicitor for Black Lung Benefits. His address is Frances Perkins Building, Room N-2605, 200 Constitution Avenue, NW, Washington, D.C. 20210.